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THE SURGICAL CLINICS OF NORTH AMERICA

FEBRUARY 1973

SURGERY OF THE HEAD AND NECK

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THE SURGICAL CLINICS OF NORTH AMERICA

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HEAD AND NECK SURGERY

George E. Block, M.D.
Guest Editor

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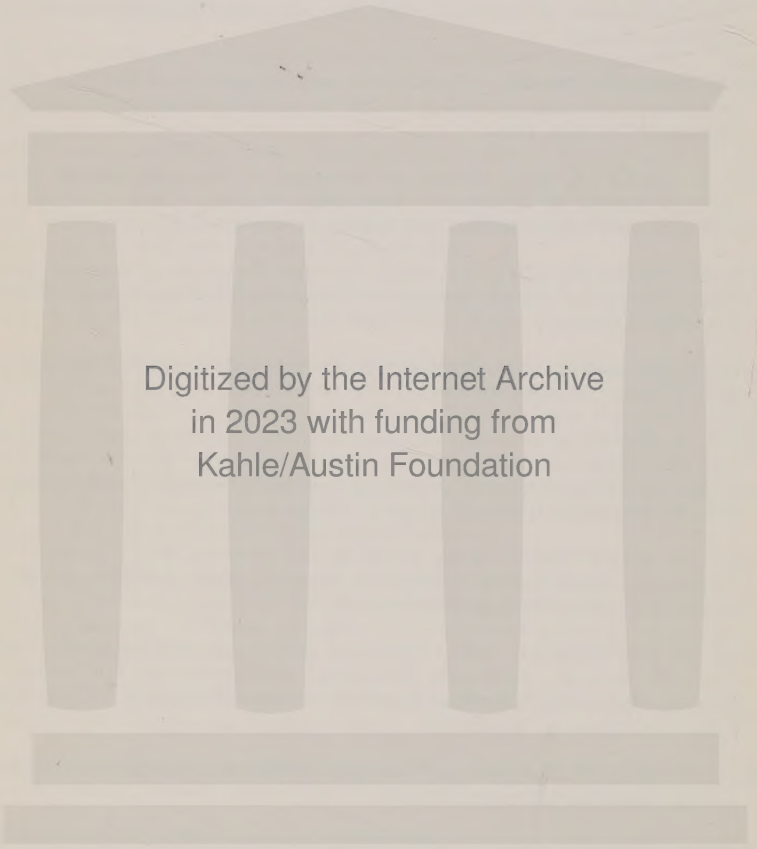
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Foreword

This issue of the *Surgical Clinics of North America* is not a syllabus of disease affecting the head and neck; nor is it an atlas of operative techniques applicable to the region. Rather, this volume is a recognition of the complex problems inherent in surgery of the head and neck and the special skills needed to solve them.

This collection of essays is intended for the general surgeon, the surgical resident, and the student of surgery. It contains the collective experience of a number of distinguished physicians and surgeons from the Chicago area—all of whom have developed expertise in a particular area concerned with diseases of the head and neck.

The plastic surgeon, the otolaryngologist, the neurosurgeon, the oral surgeon, and the radiotherapist have each developed particular skills applicable to the head and neck. We have included articles concerned with a number of these areas of special interest. Each of these articles either emphasizes a special technical skill that should be possessed by the general surgeon if he is to effectively treat lesions of the head and neck, or outlines modalities of treatment afforded only by the specialist working in concert with the primary physician.

The major portion of this volume is devoted to the modern treatment of those diseases of the head and neck frequently encountered by the general surgeon. This emphasis, coupled with sections on basic surgical skills and adjuvant therapies, will hopefully afford to the surgeon an improved ability to cope with the serious problems commonly designated as "surgery of the head and neck."

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The Management of Common Facial Fractures

*Richard Carlton Schultz, M.D.**

Man's face is his single most distinguishing physical characteristic. It is at once the key to his identity and his primary means of communicating both thought and emotion. Acknowledging these important functions of the face, modern society has come to place a premium on its preservation.[4]

Facial bones are some of the most complex bones in the body and yet they can be rather readily classified (Table 1). Treatment can then be outlined according to such a classification.

TRIAGE IN TREATMENT OF FACIAL INJURIES

In the management of patients with facial fractures, it is important to put these fractures in perspective relative to priorities in treating the whole patient.[8] While facial injuries must never be neglected, and indeed should receive attention of the highest professional skill, they are almost never fatal and ordinarily can be considered rather low in priority of treatment Most facial fractures can wait up to 2 weeks for definitive treatment, without jeopardizing the functional and esthetic result. Because of the resiliency of the facial bones of children, fractures are uncommon and tend to be greenstick in type when they do occur. Since bony union begins promptly in children, reduction should be performed as soon as possible, preferably within 1 week after injury, to avoid mal-union.

Fractures of the nose, zygoma, zygomatic arch, maxilla, and orbital areas are not usually painful and show no tendency to progressive displacement following the initial fracturing force. Mandibular fractures, however, are often painful and may become progressively displaced through muscle contraction.

A clinical relationship has been found between the prominence of certain facial bones and their vulnerability to fracture.[5] The nasal bones, which are the most prominent bones of the face, are fractured with far

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Table 1. *Classification of Facial Fractures*

Type of Fracture
Closed (simple)
Open (compound)
Greenstick
Comminuted
Undisplaced
Displaced
Location of Fracture
Upper face
Frontal bone
Frontal sinuses (glabella)
Supraorbital ridge
Middle third of face
Nasal bones and septum
Maxillary sinuses (antrum)
Orbital bones
Zygoma
Zygomatic arch
Maxilla
Transverse (Le Fort I)
Pyramidal (Le Fort II)
Cranial facial disjunction (Le Fort III)
Alveolar process
Maxillary dentition
Lower third of face (Mandible)
Mandibular dentition
Alveolar process
Symphysis
Corpus or body
Angle
Ascending ramus
Condyle
Coronoid process

greater frequency than any other facial bones. The next most commonly fractured bone is the cheek bone, or zygoma. Interestingly, the maxilla and the mandible have approximately the same incidence of fractures, but this may be a reflection of the etiology of current types of injuries.[6] Most facial fractures treated today are a result of automobile accidents.

There are definite points of weakness in the facial bones and fracture through these areas should be anticipated.[4] In addition, facial bones that demonstrate hypogenesis or congenital malformations, jaws in malocclusion, and areas of severe dental caries all have higher incidence of fracture.

Because serious injury to the skull and brain deserves prime consideration, concomitant facial injuries are managed by the simplest possible method until the patient's general condition has stabilized. However, skull fractures in themselves are not a contraindication to reduction of facial fractures.[4] Tolerance for general anesthesia is of more serious concern than facial bone manipulation. In fact, cerebrospinal rhinorrhea sometimes may stop following reduction of displaced fractures about the midface and glabellar regions.

DIAGNOSIS OF FACIAL FRACTURES

Facial fractures can be diagnosed by observation, palpation, and x-ray examination.

Observation begins with evaluation of the patient as a whole, evaluation of pain, and gross comparison of the two sides of the face. Localized swelling or ecchymosis should arouse suspicion of underlying fracture. Symmetry is an important observation. Although the uninjured face is seldom exactly symmetrical, asymmetry secondary to trauma is usually obvious.

Palpation reinforces observation. This should be performed in a systematic bilateral manner to avoid missing more subtle deformities. Beginning superiorly, at least the following points should be noted.

1. supraorbital and lateral orbital rims
2. inferior orbital rims
3. malar eminences (zygoma)
4. zygomatic arches
5. nasal bones
6. maxilla
7. mandible

X-ray examination usually merely confirms a diagnosis established by observation and palpation. Gross facial fractures can ordinarily be diagnosed without x-ray confirmation, yet there are some grossly displaced facial bone fractures which will not visualize well on x-ray. Nevertheless, such studies serve a definite function in the evaluation of facial injury. In any medical report, documentation of a fracture by x-ray serves many purposes, especially in regard to future review of the patient's injuries. The most informative views are:

1. Waters' (occipitomental) view
2. Posteroanterior view of facial bones
3. Lateral view of facial bones
4. Lateral view of nasal bones
5. Occlusal view of nasal bones
6. Posteroanterior view of mandible
7. Oblique view of mandible
8. Occlusal view of mandible
9. Townes view (for ascending mandibular rami and condyles)
10. Tangential views of zygomatic arch

The single most informative x-ray view of the facial bones is the Waters' (occipitomental) view. This film is taken with the patient in the prone position with the neck somewhat extended, and not all patients, especially those having sustained multiple areas of injury, can assume this position. Though not as clear or as informative, a substitute view – the reverse Waters' – can be taken in the supine position.

More precise or additional information can sometimes be derived by the use of special x-ray techniques. Among these are laminographic studies (tomography) and panoramic (Panorex) scanography. The latter is less commonly found in hospitals but is popular in dental offices. It is especially valuable in demonstrating hard to visualize fractures of the mandible, particularly in the condylar areas.[4]

ZONES OF FACIAL BONES

To simplify the description of facial bones we can refer to the zones of the face outlined above: the upper third of the face, the middle third of the face, and the lower third (Fig. 1). Before briefly describing treatment of typical fractures in each of these zones, the nature of facial bone construction should be considered. Facial bones are generally thin and relatively unsupported. They form a compressible, energy-absorbing shield for those vital organs which lie within and behind them. These specialized organs—the eye and the lacrimal apparatus, the pharynx, the cervical spine, and the brain—must be of prime concern to any physician treating facial injuries.

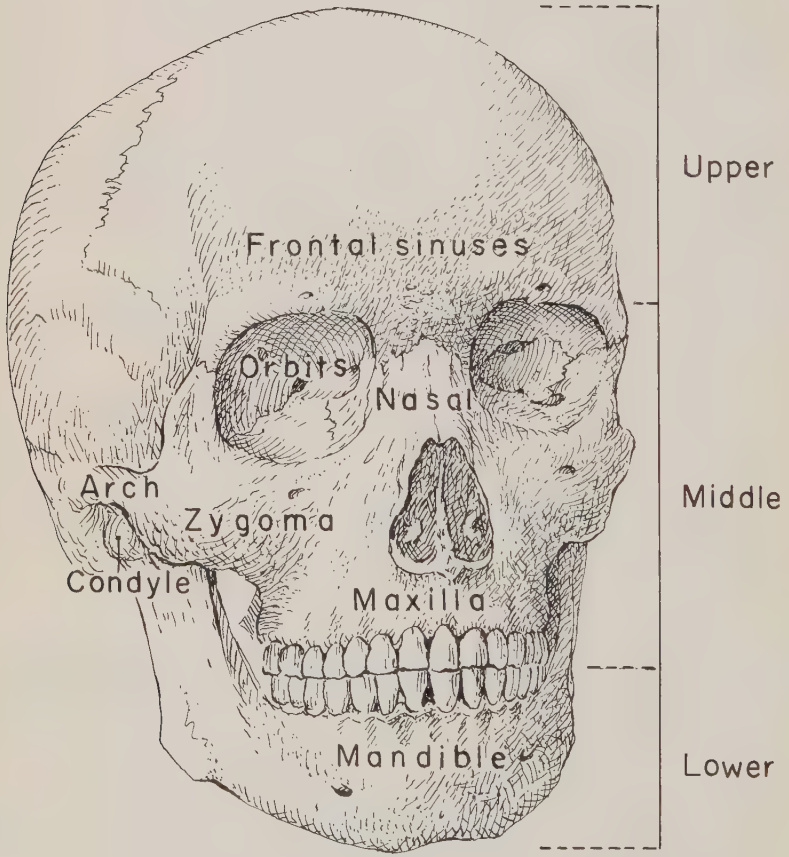


Figure 1. Area identification of facial bones.

FRACTURES OF UPPER THIRD OF THE FACE

Fractures of this zone of the face are infrequent but are potentially the most serious of all facial bone fractures because of the possibility of underlying cerebral injury. Patients with such injuries have the longest hospital stay of all patients with facial injury.[7] These fractures are most commonly sustained in automobile and motorcycle accidents.[9] The bones most vulnerable to injury in the upper third of the face are the frontal sinuses and that portion of the frontal bone designated as the supraorbital ridge.

Diagnosis. Initially glabellar and supraorbital depression may be masked by overlying swelling. Tenderness and crepitus can usually be elicited by palpation. However, x-ray films best demonstrate such fractures on postero-anterior, lateral, and Waters' projections, although some of the fractures are difficult to visualize in any view.

Treatment. Because of the difficulty in determining the complexity of both glabellar and supraorbital fractures by physical x-ray and examination, surgical exploration is usually indicated. Access to these fractures can be gained through an overlying soft tissue wound or through one of several planned incisions. The normal inferior horizontal skin line of the forehead may be used, or an incision may be made just superior to each brow and connected across the midline just above the nasal bridge (Fig. 2).

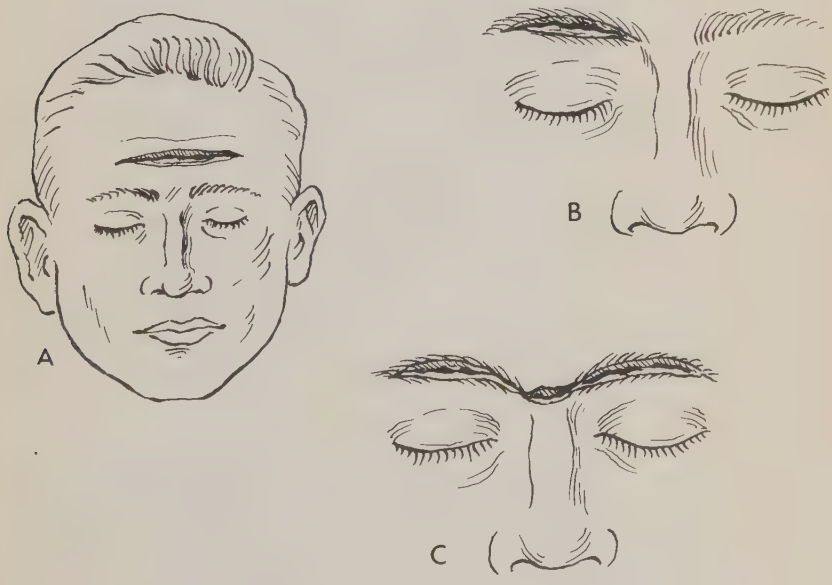


Figure 2. Elective incisions for exposure of glabellar and supraorbital fractures.

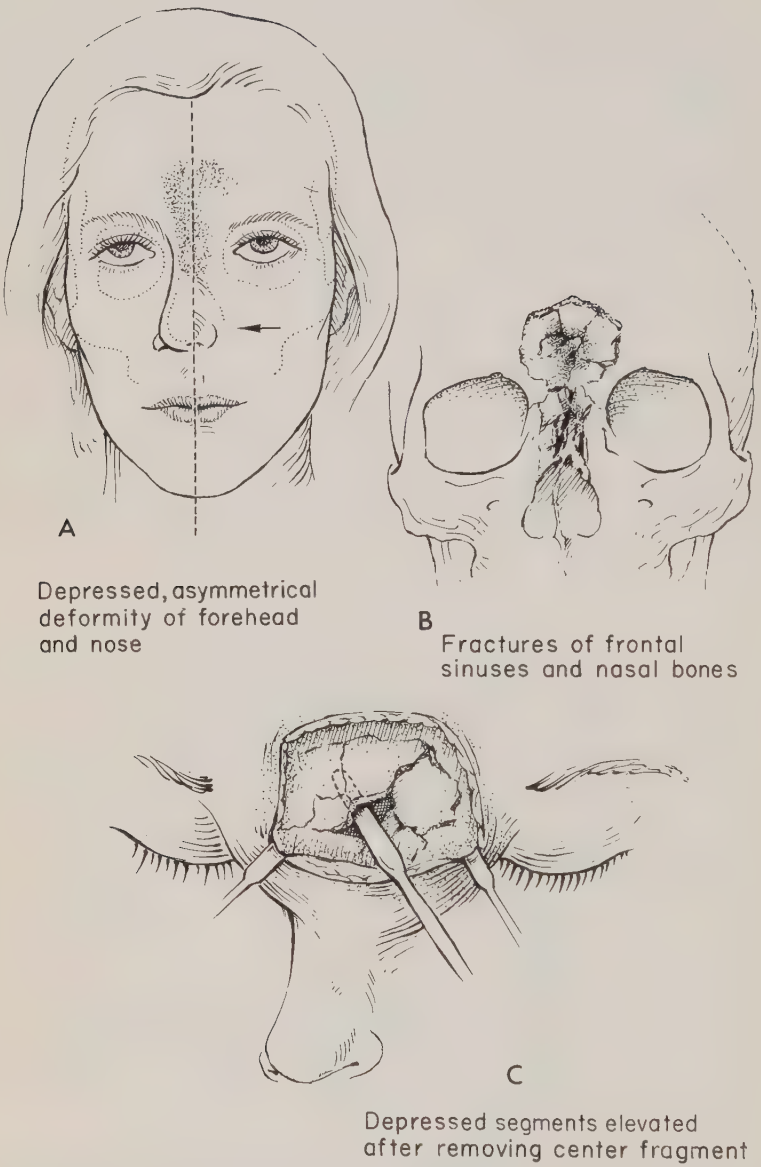

A
Depressed, asymmetrical deformity of forehead and nose
B
Fractures of frontal sinuses and nasal bones
C
Depressed segments elevated after removing center fragment

Figure 3. *A* and *B*, Depressed fractures of frontal sinuses and nose. C, Technique for elevating depressed fracture of frontal sinus after removal of central fragment.

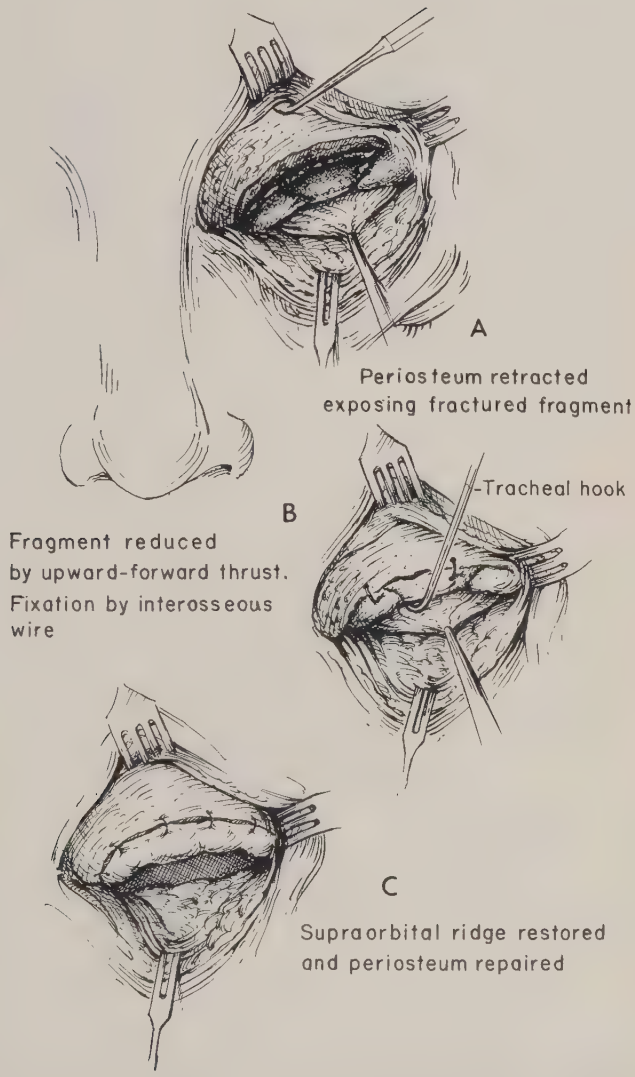


Figure 4. Operative technique for reduction of comminuted supraorbital fractures with tracheal hook.

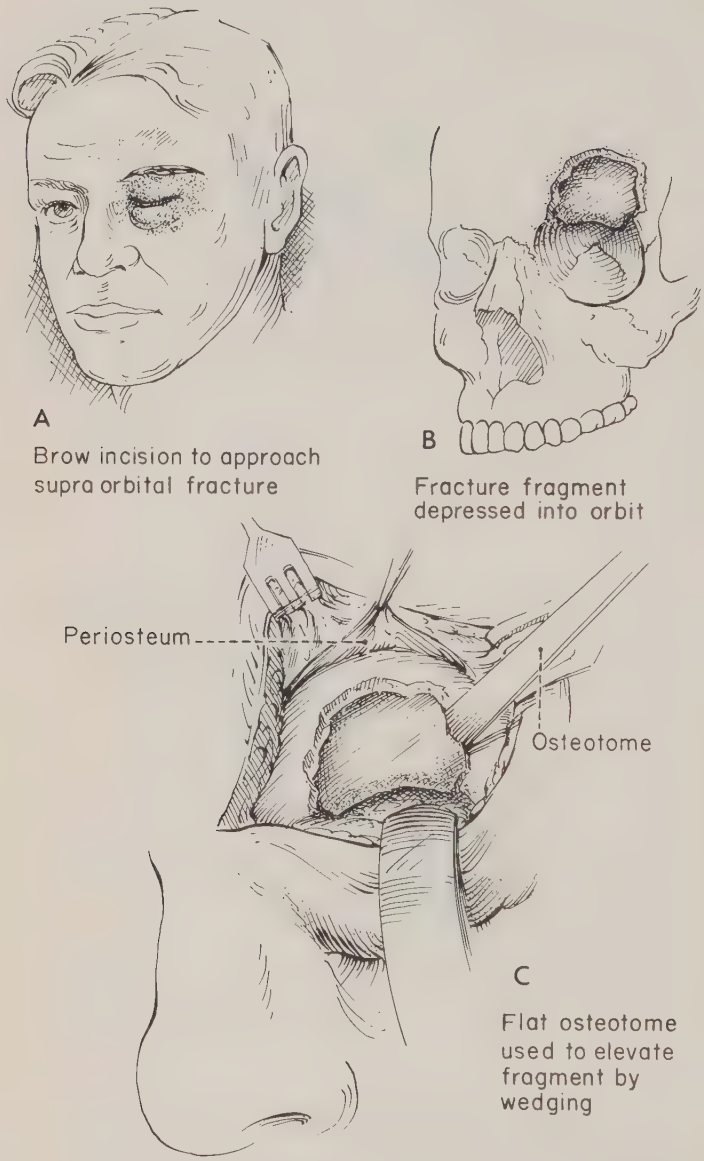


Figure 5. Operative technique for reduction of depressed supraorbital fracture by wedging maneuver.

Depressed bone fragments must be elevated delicately, preserving any attachments between the fragments and where possible preserving periosteal contact with the fragments. If there is a small access to the frontal sinus a thin curved periosteal elevator can be inserted into the cavity. By gentle force the depression can sometimes be corrected and maintained simply by re-establishment of form and periosteal contact (Fig. 3). Similarly, a depressed supraorbital ridge can often be elevated by gentle upward-forward force with a tracheal hook (Fig. 4) or wedging with a flat thin osteotome (Fig. 5). When these fragments do not remain stable in reduced position, they must be wired to adjacent stable bone.

Resection of the frontal sinuses and lining membranes is sometimes advocated as a form of management in crush-type injuries.[1,2,10] Although such a practice does solve the immediate problem, the invariable result is a depressed glabellar deformity which is difficult to subsequently reconstruct satisfactorily. [7]

FRACTURES OF MIDDLE THIRD OF THE FACE

The most common facial fracture and, ordinarily, the simplest to treat—the nasal fracture—is found in the middle third of the face. Conversely, the most demanding—the maxillary fractures—lie within the same middle third zone. Since this portion of the face is the key area for both function and appearance, fractures here deserve attention of the highest order. It is in this zone that the greatest number of fractures are missed and the greatest mistakes in judgment are made relative to treatment. The bones making up the middle third of the face are thin and lie in numerous planes. With denser cranial bones behind them, even grossly displaced fractures may be difficult to demonstrate by x-ray examination. For these reasons, diagnosis of fractures in this zone are best made by physical examination.

Nasal Bones

Although usually obvious, the diagnosis of nasal fracture can be rather perplexing when displacement is minimal. In such cases re-examination after resorption of swelling is helpful in determining whether or not reduction is necessary. As a general rule, if x-rays suggest displacement, reduction should be scheduled since more subtle deformities can be masked by edema when the patient is examined shortly after the injury.

Diagnosis. Regional swelling, tenderness, periorbital ecchymosis, deformity or angulation, and crepitus are possible findings on physical examination. Routine facial x-rays often do not demonstrate nasal fractures. Lateral and occlusal views of the nasal bones specifically should be ordered if a nasal fracture is suspected.

Treatment. Truly undisplaced fractures (not just radiographically undisplaced), do not require treatment. All other acute nasal fractures should be treated by closed reduction, and nearly all can be done under local anesthesia.

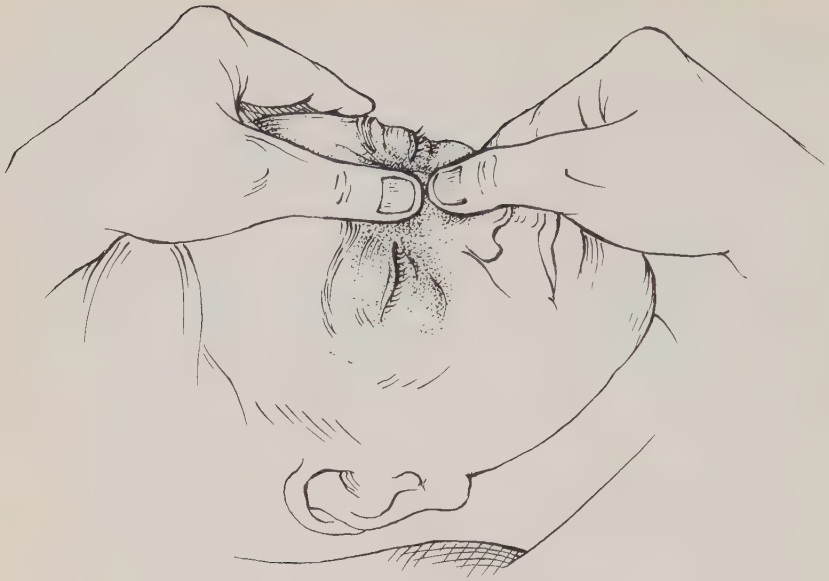


Figure 6. Manual reduction of nasal fracture.

Acute angulated fractures, when seen early before there is significant swelling, can sometimes be reduced by manual pressure with or without anesthesia. A quick push of the bony pyramid toward the midline with both thumbs (Fig. 6) can sometimes achieve anatomic reduction with only momentary discomfort to the patient.

Most other nasal fractures are best reduced under sedation and local anesthesia. Cocaine is still probably the best topical anesthetic agent available for nasal mucous membranes. Five ml. of a 10 per cent cocaine solution mixed with equal parts of epinephrine (1:1,000) will quickly provide complete topical anesthesia and at the same time cause sufficient local vasoconstriction to minimize bleeding related to surgery. The cocaine solution can be easily applied with cotton dental rolls (Fig. 7). After packing the nose, an external block is performed by injection of local anesthesia (lidocaine with epinephrine) (Fig. 8).

Once regional block anesthesia has been achieved, the fracture can be manipulated with as much force as necessary to achieve reduction. Nearly all unhealed fractures can be easily reduced by manipulation with three special instruments (Fig. 9). Reduction is ascertained by observation and palpation of the nose and check x-rays postoperatively are almost never necessary. Immediately following reduction a splint fashioned of plaster of paris is shaped to exact contour of the nose (Fig. 10). Such a splint serves to minimize post-reduction swelling and protects the nose. It is easily fashioned from four thicknesses of 3 inch plaster, cut with a scissors while dry and applied wet directly over the nasal skin. Excess water is expressed while the splint is molded over the nose. Final trimming should be done while the plaster is still moist. It is easily fixed to the face with micropore tape.

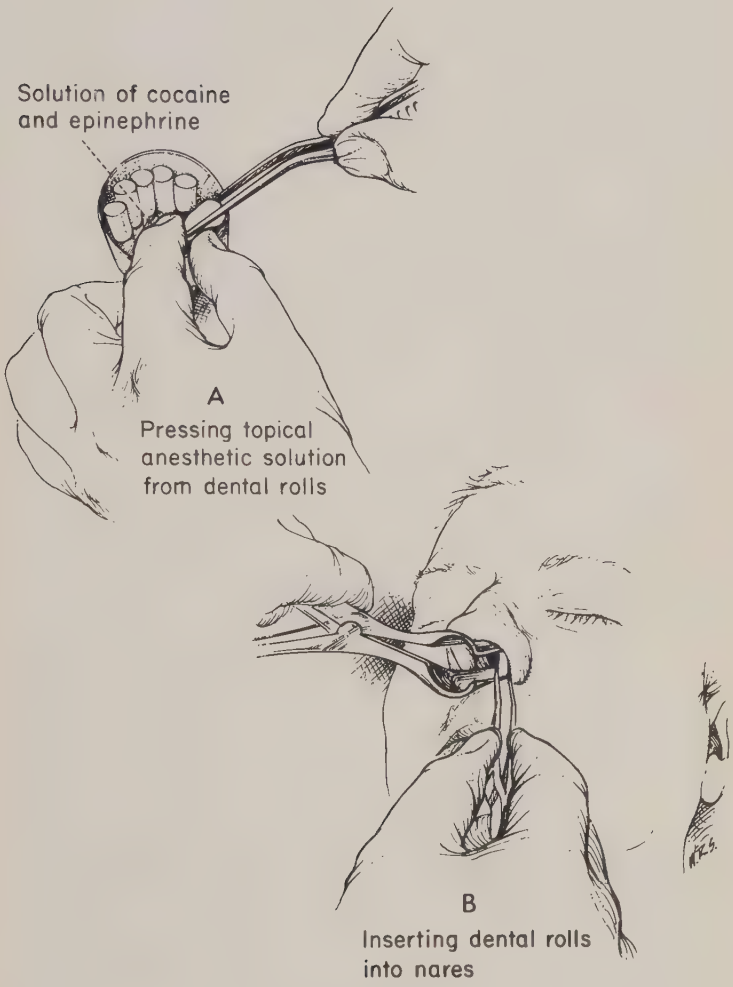


Figure 7. Technique for anesthetizing nasal mucous membranes.

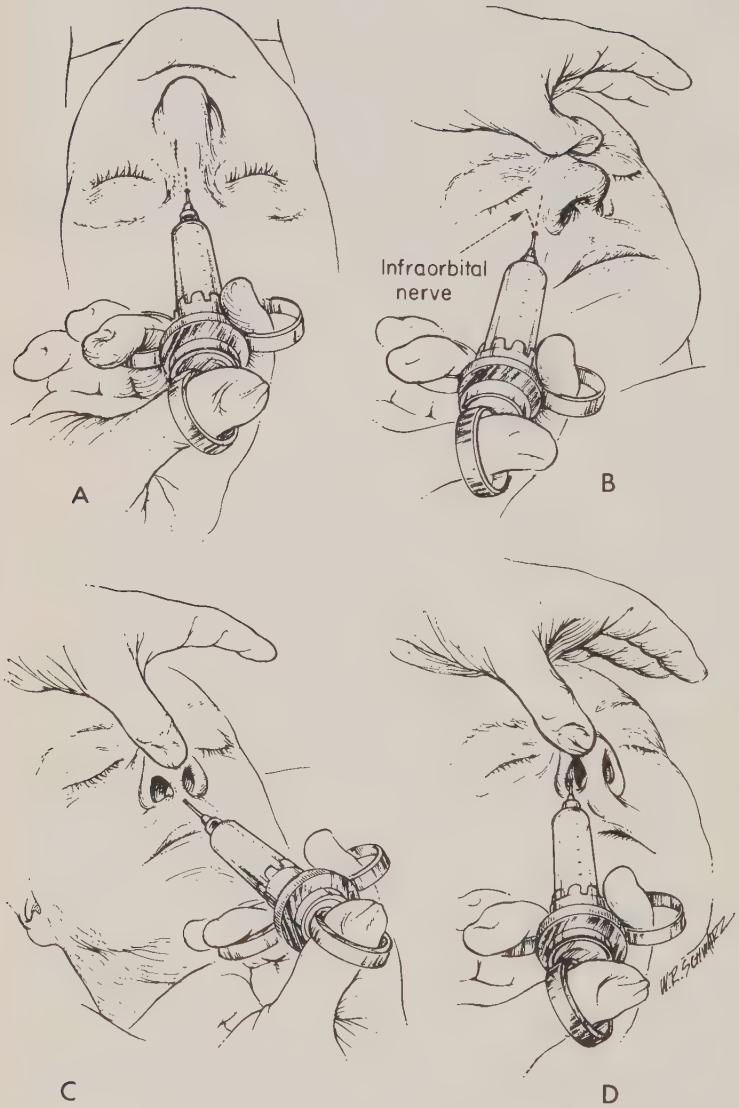
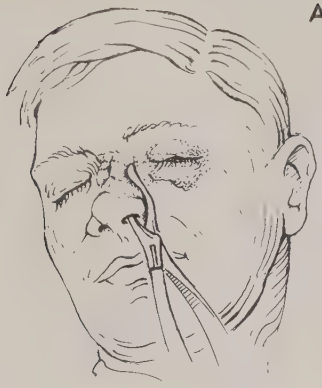
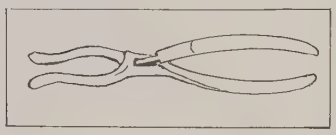


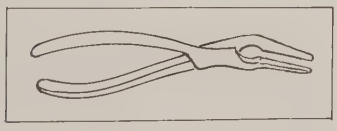
Figure 8. Technique for external nasal block.



A Walsham forceps used to disimpact and reconstruct nasal pyramid



Asch forceps used to disimpact and replace nasal septum in midline



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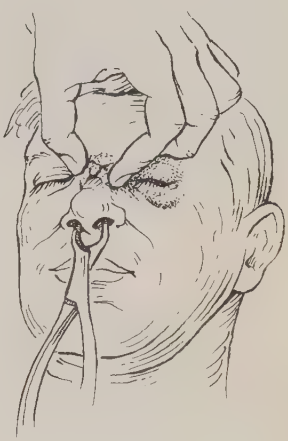
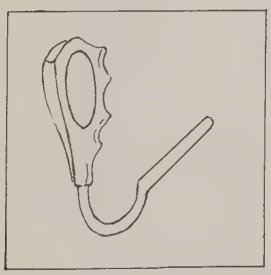


Salinger reduction instrument used to further shape and contour nasal pyramid and reduce fractures of nasal tip



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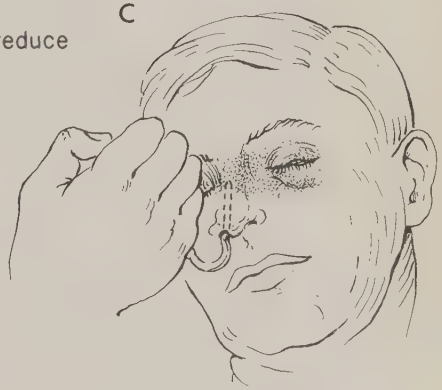


Figure 9. Instruments for reduction of nasal fractures and technique of use.

Figure 10. *Left*, Plaster of paris nasal splint tailored to patient's features. *Right*, splint fixed to face with micropore tape.

Zygoma

The zygoma, or cheek bone, is the second most frequently fractured facial bone, and fractures of the orbital rims and orbital floor commonly occur in association.

Diagnosis. The most consistent finding is periorbital ecchymosis. Other physical findings are: depression of the malar eminence, depression of the inferior orbital rim, hypesthesia in the distribution of the infraorbital nerve, and tenderness about the inferior and lateral orbital rims. Diplopia is seldom an early complaint, even when there are associated depressed fractures of the orbital floor. This is because of compensating edema in surrounding soft tissues.

X-rays demonstrate fractures of the zygoma and orbital rims best in the Waters' view. In addition to visualizing the fractures themselves, a consistent finding is clouding of the maxillary antrum. Comminution with depression of the orbital floor is common, but difficult to demonstrate except by laminographic studies.

Treatment. Depressed fractures of the zygoma are best treated by open reduction and internal wire fixation. To do less invites late depression deformity of the zygoma and enophthalmos. If an associated depressed fracture of the orbital floor has been left untreated, diplopia may appear as a late symptom.

An open reduction can be best accomplished through two incisions: one just beneath the palpebral margin of the lower eyelid and the second across the lateral orbital rim, beneath or in the lateral aspect of the eyebrow (Fig. 11*A–D*).

Only after elevation of the periosteum at the fracture sites can the

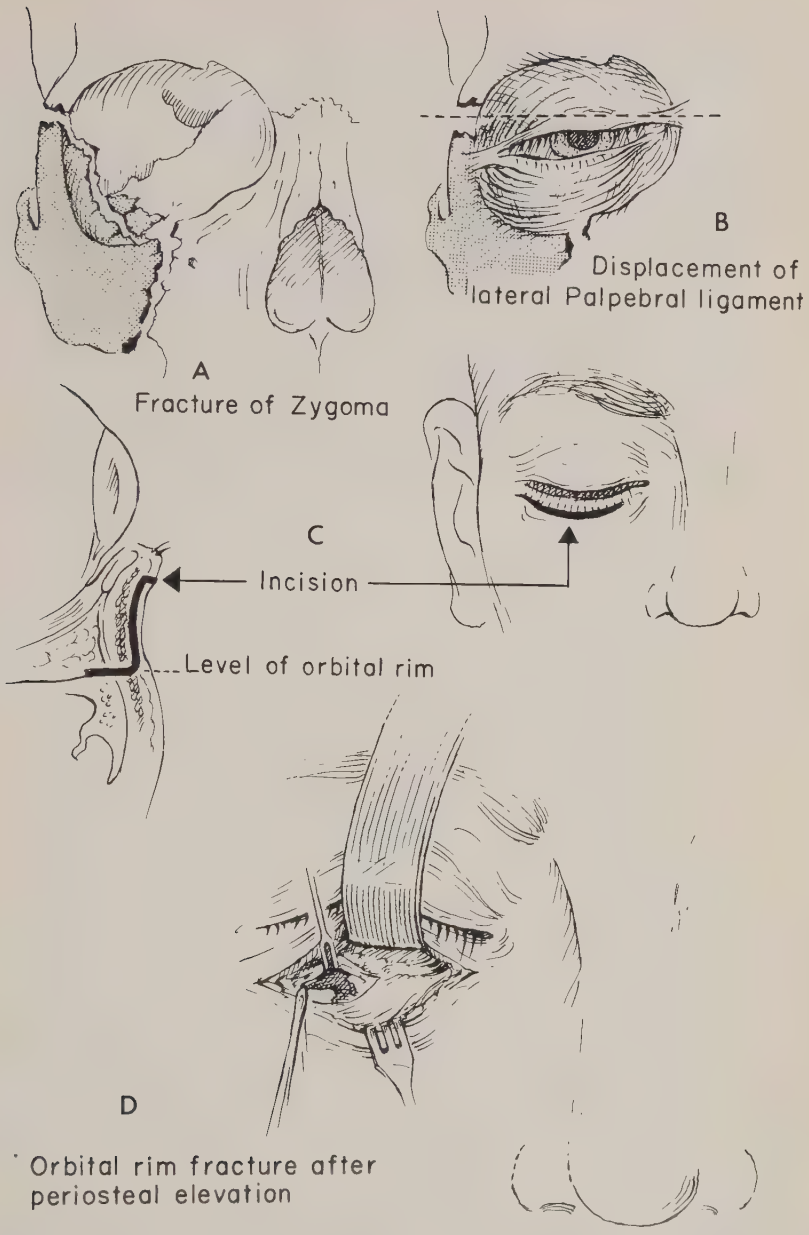
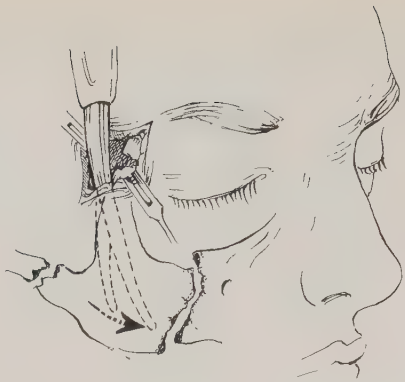


Figure 11. *A* to *D*, Operative technique for open reduction and internal wire fixation of zygoma fractures.

(*Figure continued on following pages.*)



E

Elevation of Zygoma by upward-forward force

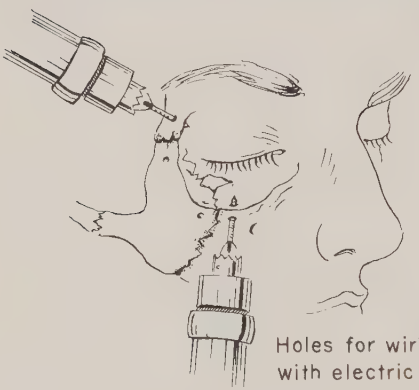


Figure 11 (*Continued*). *E* and *F*, Reduction of zygoma and drilling holes for wire fixation.

degree of depression of the zygoma and orbital floor be determined. After reduction of the fractures and wiring at the inferior and lateral orbital rims when necessary (Fig. 11*E* and *F*), some attention should be directed to the orbital floor. When comminution is found, the floor should be supported to prevent subsequent depression and enophthalmos. The simplest and yet most adequate way to accomplish this support is to insert a thin (0.02 to 0.04 inch) sheet of alloplastic material (Silastic), shaped to fit the floor of the orbit, beneath the periosteum. If the implant is shaped so that it will lie flat and the anterior edge sits behind the edge of the orbital rim (Fig. 11*G* and *H*), it is not necessary to fix the implant other than to close the enveloping periosteum.

Maxilla

Most facial fractures today are sustained in automobile accidents.[6] Considering this etiology, fractures of the maxilla and fractures of the

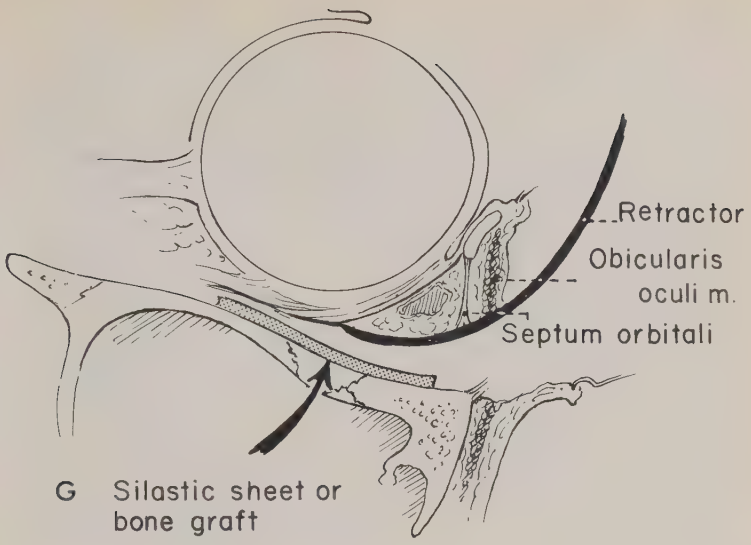


Figure 11 (*Concluded*). *G* and *H*, Implant used to reinforce orbital floor.

mandible occur in approximately the same frequency. Fractures of the maxilla often go unrecognized in the cursory physical examination and routine skull x-rays obtained in a busy emergency room. Maxillary fractures occur with great variety but the major variations can be best understood by reviewing the original descriptions of Le Fort:[5] I, transverse; II, pyramidal; and III, craniofacial disjunction (Fig. 12).

Diagnosis. Maxillary fractures can all be diagnosed by *careful*

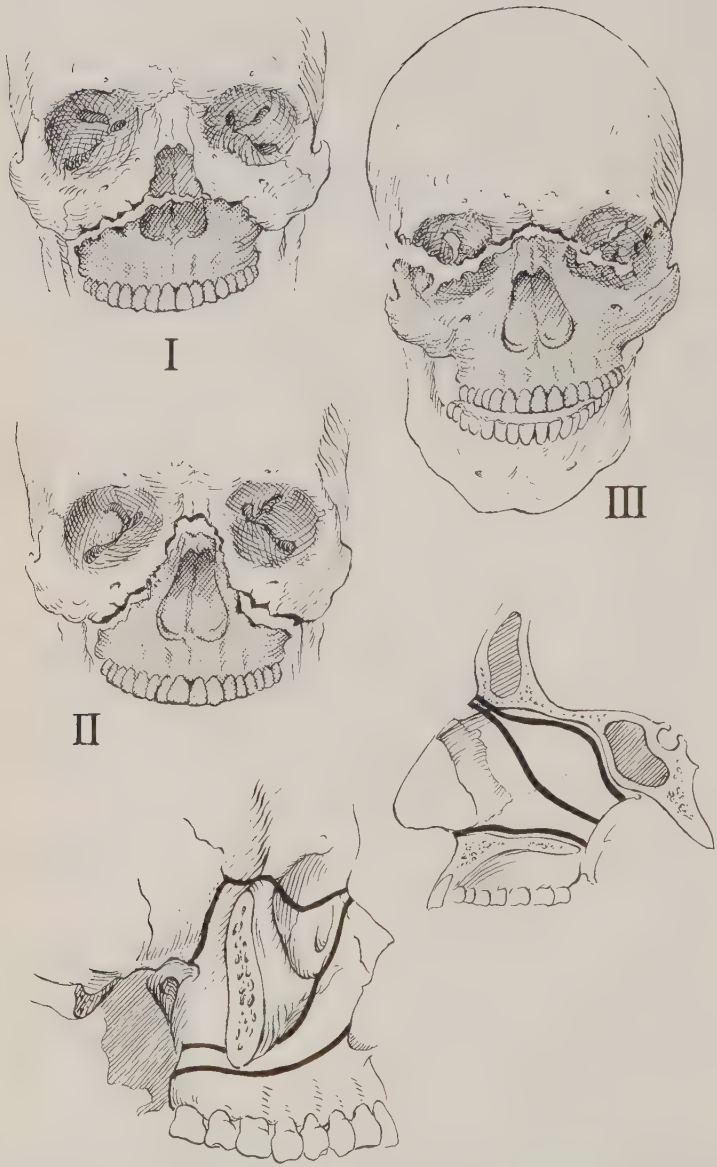


Figure 12. Le Fort classification of maxillary fractures. I, transverse fracture of maxilla; II, pyramidal fracture of maxilla, III, craniofacial disjunction.

Figure 13. Anterior and lateral open bite typical of displaced maxillary fracture.

physical examination. The most consistent finding is malocclusion (Fig. 13). The most striking finding is elongation of the midface. Periorbital swelling and ecchymosis may be present but often there is no significant facial swelling. Motion of the fractured maxilla may often be demonstrated by grasping the upper dental arch. Discomfort is usually elicited.

The Waters' projection is the most informative x-ray projection for fractures of the maxilla, but laminographic studies are often necessary to demonstrate the fracture sites, especially about the orbits.

Treatment. The Le Fort classification is useful in planning surgical treatment, but variations of maxillary fractures are so common that treatment needs to be individualized more for these fractures than any other.

A well planned orotracheal intubation for anesthesia will allow access both to the midface for open surgery and to the teeth for application of archbars. Intermaxillary fixation can then be accomplished the following day without anesthesia. In this way the mouth can be left open in the immediate postoperative period, in the event of vomiting, yet the matter of intermaxillary fixation is effectively resolved.

TRANSVERSE MAXILLARY FRACTURES (LE FORT I). When vertical displacement is minimal simple intermaxillary elastic fixation is sufficient to achieve the best possible dental occlusion. Maxillary suspension is indicated when there is significant downward or lateral displacement of the fracture fragments. This is accomplished by use of wires bilaterally to suspend the upper dental arch from the temporal bone, usually at the zygomaticotemporal suture area (Fig. 14).

PYRAMIDAL FRACTURES (LE FORT II). These fractures and fractures of the zygoma have in common involvement of the maxillary antrum, the inferior orbital rims and the orbital floors. In maxillary fractures, howev-

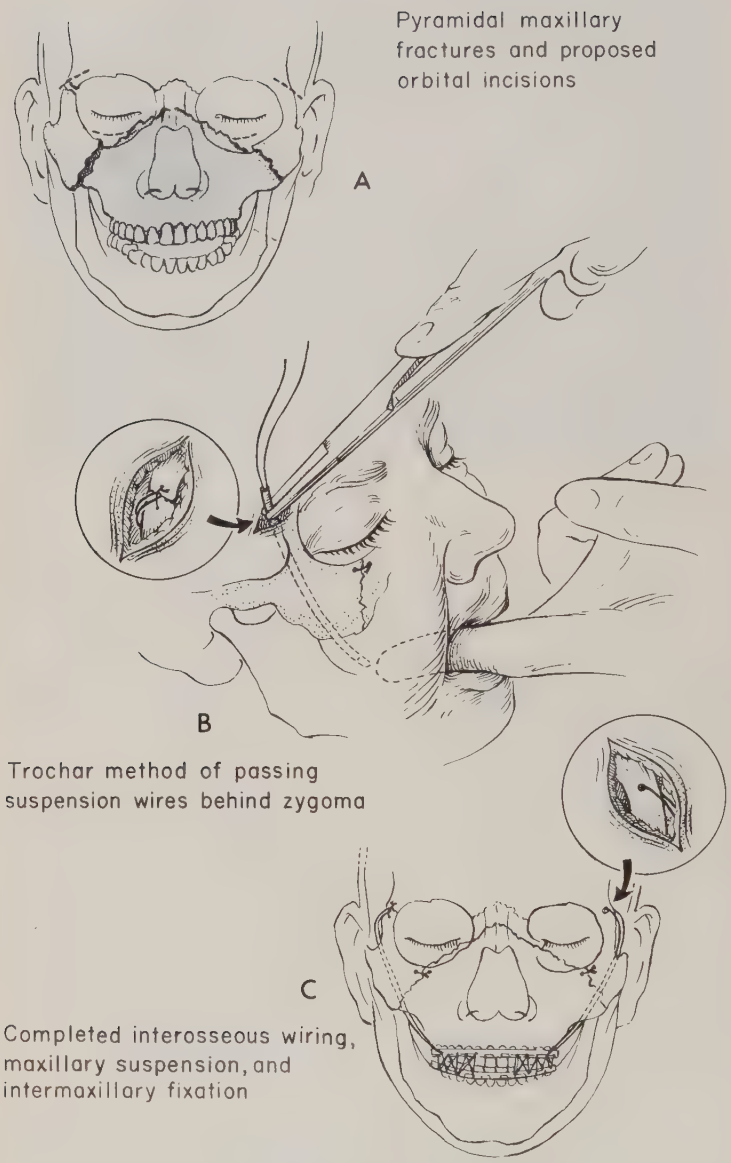


Figure 14. Technique for wire suspension of maxilla from temporal bones bilaterally.

Figure 15. *A*, Patient with pyramidal fracture of maxilla and associated depressed fractures of left zygoma and orbital floor, as well as comminuted nasal fractures. Note giant hematoma of cheek. *B*, Posterior-anterior laminagraph of facial bones, showing markedly depressed fracture of left zygoma and orbital floor. *C*, Postoperative Waters projection shows maxilla impaled by Kirschner wires and suspended from intact right zygoma. Note internal wire fixation of fractures about left orbit. *D*, Six months after operation. Left pupil is still somewhat lower than right, owing to bone absorption at comminuted fracture sites, but patient did not experience diplopia. Esthetic appearance is correctible by iliac bone graft.

er, it is the central segment that is displaced posteriorly with upward angulation so that the patient has an anterior open bite. The apex of this fracture segment involves the nasal bones and often the bones making up the medial aspect of the orbital floor.

The surgical approach is also similar to an open reduction of a fractured zygoma (Fig. 8), with internal wire fixation at the inferior orbital rims. Closed reduction of the nasal fracture is necessary to align the apex of this pyramidal segment. As with transverse maxillary fractures, when significant displacement or mobility of fracture fragments is present, the maxilla must be suspended from intact temporal bone (Fig. 11).

When there is comminution of the pyramidal portion of the maxilla and one or both zygomas are intact, the central segment can be immobilized by impaling the maxilla with Kirschner wires passed through the zygoma bilaterally. Reduction in this manner is never as accurate as with the first technique described, but in some injuries it is the best that can be done (Fig. 15).

CRANIOFACIAL DISJUNCTION (LE FORT III). This is the most complex of all facial fractures. As the name describes, it constitutes a separation of the midface from the cranium. The entire middle third of the face is fractured, including the maxilla, one or both zygomas, and the nose. Patients with this injury present a bloated, edematous midface which in itself is rather typical. A "free-floating" maxilla is found. Malocclusion due to a tilting upward and backward of the detached maxilla is seen and presents as an anterior open bite.

Although numerous special instruments have been developed for the treatment of these fractures, and special metal or plaster head caps devised for external support, the preferable treatment of almost all maxillary fractures is by open reduction and internal wire fixation,[4] following the techniques previously described. To accomplish this, each possible point of access to the fractures must be considered and appropriate reduction and fixation carried out through each one. When teeth are present, they should always be placed in the best possible occlusion and held in intermaxillary fixation. There can be exceedingly complex fractures in this category which will tax the ingenuity and skill of the best surgeons to achieve a good result.

FRACTURES OF LOWER THIRD OF THE FACE

Mandible

Unlike most other facial fractures, mandibular fractures can be painful because of the distracting forces of attached muscles (Fig. 16).

Diagnosis. The most consistent physical finding in mandibular fractures, as in fractures of the upper jaw, is dental malocclusion. Whereas an anterior open bite is most often seen with maxillary fractures, lateral crossbite is most often seen with mandibular fractures. Other physical findings are related to the exact location of the fracture (see Table 1 and Fig. 17). Deformity, arch collapse, mobility of fracture segments, swelling and ecchymosis, lingual deviation of the teeth, and tenderness elicited by palpation are all common findings. X-rays are helpful in demon-

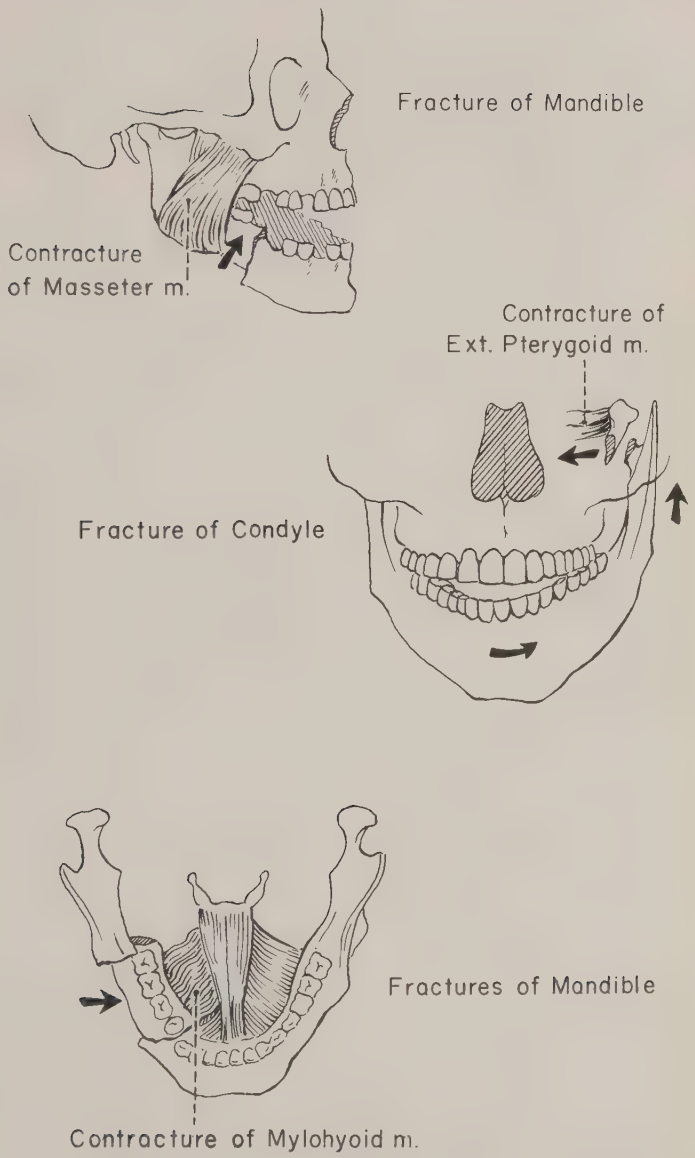


Figure 16. Muscles which tend to distract mandibular fracture segments.

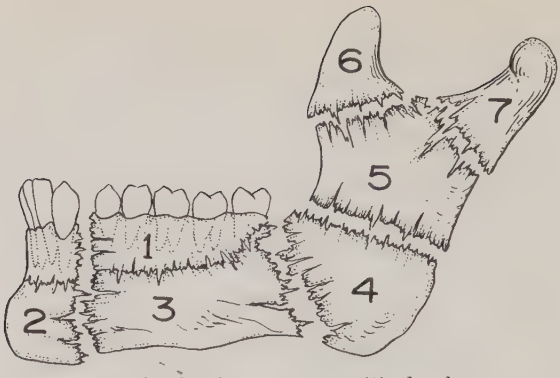


Figure 17. Common mandibular fracture sites: (1) alveolar process; (2) symphysis; (3) body; (4) angle; (5) ascending ramus; (6) coronoid process; (7) condyle.

strating the exact location and nature of mandibular fractures. The most useful projections are: lateral or oblique, posterior-anterior, Townes, and occlusal. Laminography is not particularly useful except for the condyle and temporomandibular joint area and here Panorex may be even more helpful.

Treatment. Aims of treatment are to control pain and achieve bone healing with the best possible dental occlusion in the shortest time. In fulfilling these aims, appearance and symmetry are restored. With undisplaced asymptomatic fractures, treatment consists of periodic observation and mechanically soft diet. Most fractures, however, require some form of interdental, intermaxillary, or interosseous fixation.

Interdental Fixation. Fractures of the alveolar process, and sometimes transverse mandibular fractures can be treated by manual reduction and interdental fixation alone (Fig. 18).

Intermaxillary Fixation. Displaced fractures of the mandible with teeth present almost always require intermaxillary fixation to obtain the best possible occlusion. There are two simple techniques for this which will immobilize satisfactorily almost any mandibular fracture. These are the Blair Ivy (eyelet) loops and the archbar fixation (Figs. 19 and 20).

In ligating the teeth with wire, the molar teeth serve best for anchorage. The canine and premolar teeth can be used but the incisors should always be avoided because of their unstable roots.

Once the hardware is secured to the maxillary and mandibular teeth, the jaws are brought together by either small elastics or additional wire. Elastics are always used when complete reduction is not possible manually; otherwise, they are used by choice. They have the advantage of being more easily adjusted or replaced, and they may be quickly cut in the event of vomiting. Wires are useful in maintaining reduction in the uncooperative patient and they permit easier intraoral hygiene.

Mandibular fractures usually require about 6 weeks of immobilization for clinical healing. Despite lack of x-ray evidence of healing at the end of that time, the intermaxillary fixation (either elastics or wire) may be removed leaving the Blair Ivy Loops or archbar in place. If the fracture site seems solid clinically the patient is started on a soft diet and re-

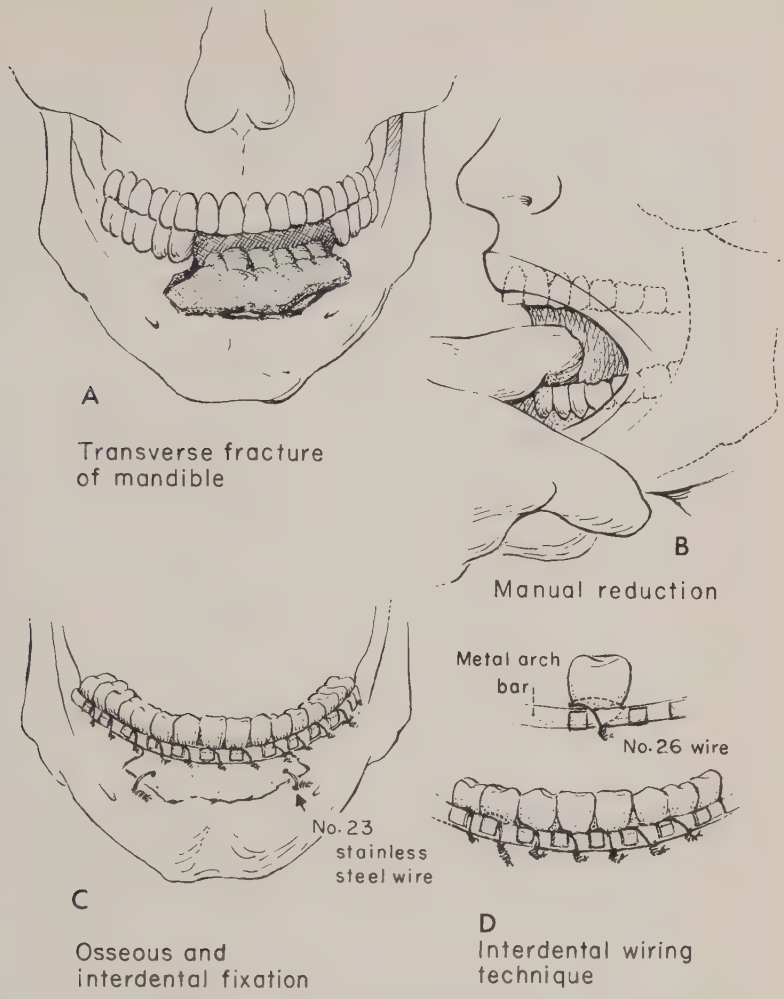
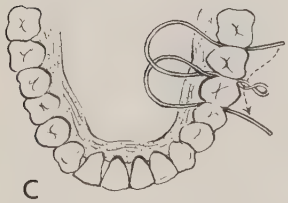
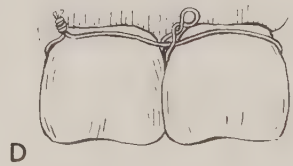


Figure 18. Manual reduction and fixation methods for transverse mandibular fractures.

evaluated in one week. If pain or motion returns at the fracture site in this 1 week interval, the intermaxillary fixation should be reapplied for an additional 2 weeks. Otherwise the wires can be cut and either the loops or the archbars can be removed. This can be done in the office without anesthesia.

Interosseous Fixation. Fractures of the angle or multiple mandibular fractures may prove unstable with intermaxillary fixation alone and require interosseous fixation (Fig. 21). Unstable and displaced fractures in the edentulous mandible also require open reduction and interosseous fixation (Fig. 22). In addition, some subcondylar fractures with marked displacement are best treated by open reduction and interosseous fixation. Interosseous fixation is accomplished by direct wiring of bone fragments, with Kirschner wires or with metal plate and screws. The





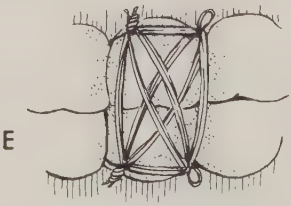
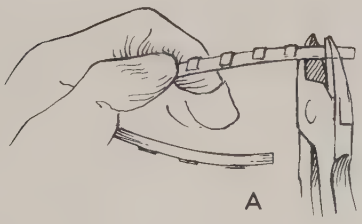
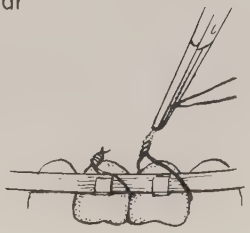


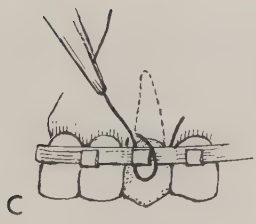
Figure 19. Formation and application of Blair Ivy Loops.



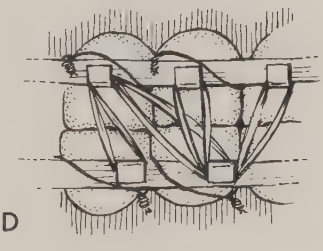
Cutting and shaping metal arch bar to fit dental arch



Simple ligation of arch bar to teeth with wire



Special technique for fixation to canine tooth



Intermaxillary fixation with dental elastics

Figure 20. Formation and application of dental arch bars.

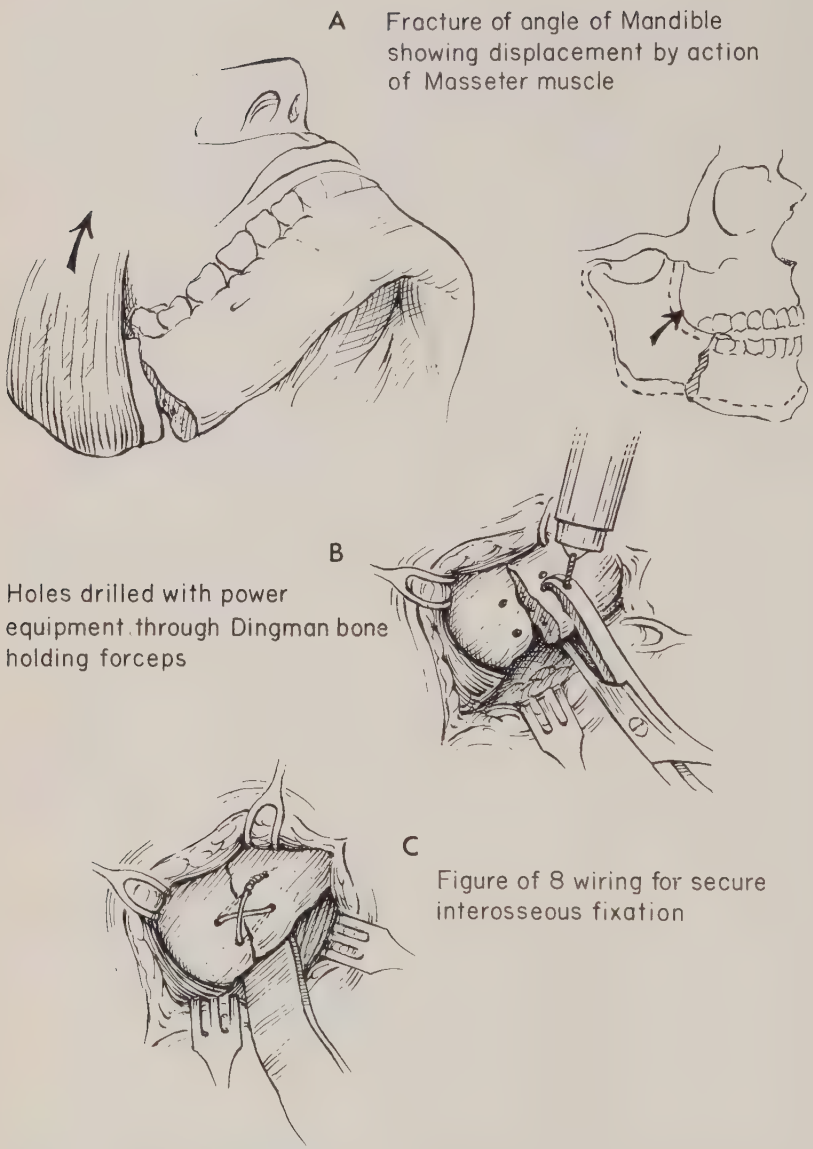

A
Fracture of angle of Mandible showing displacement by action of Masseter muscle
B
Holes drilled with power equipment through Dingman bone holding forceps
C
Figure of 8 wiring for secure interosseous fixation

Figure 21. Operative technique for open reduction and figure eight internal wire fixation of fracture at mandibular angle.

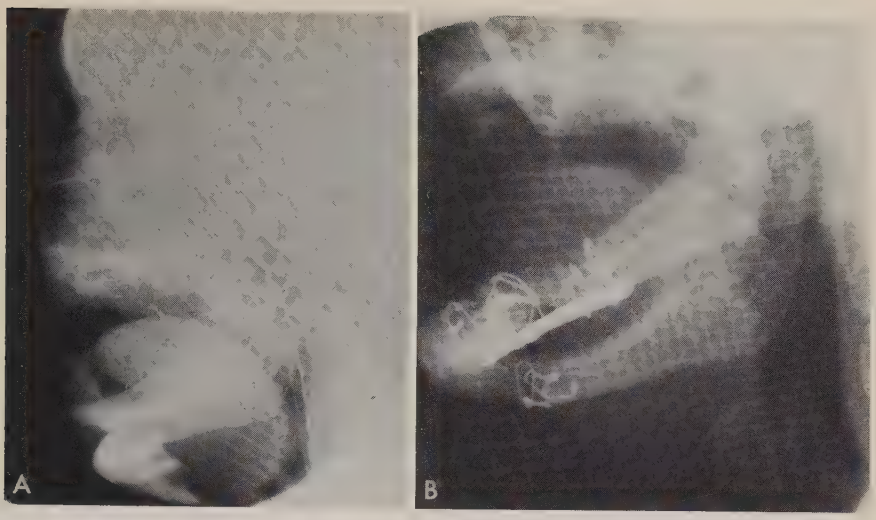


Figure 22. *A*, Markedly displaced comminuted fractures of anterior mandible in an edentulous patient. *B*, X-ray of mandible postoperatively showing reduction of multiple fractures and internal fixation using stainless steel plate and wires.

Roger-Anderson type of external fixation with pins penetrating the mandible through the skin is now rarely used. It has fallen into disfavor because of problems of continuous adjustment, osteomyelitis of the mandible, patient discomfort, unwieldiness of the apparatus, frequent mal-union and undesirable scarring of the face by the pins.

In direct interosseous wiring injury to the marginal mandibular branch of the seventh nerve can be avoided if the incision is made high on the neck and the fracture is approached beneath a thick flap of skin and platysma muscle. Wiring of the reduced fracture fragments must accomplish stability in the mandible or delayed or even non-union may result. These interosseous wires should never penetrate the oral mucosa. They can be left permanently in place after primary healing occurs. Where persistent seropurulent drainage is observed the wires must be removed. Adjunctive intermaxillary fixation will afford even greater stability and more prompt healing at the fracture site.

NOURISHMENT AND HYGIENE

Eating becomes a problem when there are fractures of the maxilla or mandible. Motion resulting from chewing (or talking) can be deleterious to fracture healing. Chewing is impossible when there is intermaxillary fixation. The ideal nourishment for these patients is a liquid diet taken by straw. There are a number of nourishing commercial products (Meretene, Carnation Instant Breakfast) available in powder and liquid form to provide all the nutriment and calories necessary to support these patients indefinitely. After several days, however, patients tend to object to these preparations because of the monotony of taste. When patients are sent

home and increase their activity, they usually crave a more varied diet. This can be provided with the aid of a modern food blender. Regular table foods can be mixed in a blender, and by adding sufficient liquid, a mixture fluid enough to pass through a straw can be obtained.

For hygienic purposes important to wound healing and to promote a feeling of well being, the teeth and mouth should be cleaned regularly. However, without special instructions and encouragement, patients with facial fractures are apt to neglect cleaning their teeth. This is especially so in patients with intermaxillary fixation. A mouthwash of half strength (1.5%) hydrogen peroxide can be swished back and forth between the teeth for a cleansing effect. Most effective of all is a pressurized warm water stream cleanser (Water Pik). The thin pulsating stream of water can be directed where debris collects, particularly about the intermaxillary fixation wires and bars. An infant's soft toothbrush can also be of great help in cleaning the teeth and fixation device in a mouth which is tender and difficult to open.

BONE DEFECTS AND DEFORMITIES

Most commonly facial bone deformities following trauma result from unreduced or inadequately reduced fractures. Occasionally bone is lost by avulsion, absorption or infection, but depressed fractures are by far the commonest cause of bone deformity. Corrective surgical procedures consist of open reduction techniques, bone grafts and foreign material implantation.[4] If necessary, these procedures can be done in combination.

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Basic Plastic Surgical Techniques in Repair of Facial Lacerations

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There is no doubt that the most common operative procedure performed by the young (and the not-so-young) surgeon is the suturing of a laceration. With the increased incidence of facial injuries secondary to automobile, industrial, and sports accidents, the young physician has a plentiful supply of clinical material. Depending on what is done, or, more important, what is not done, his initial treatment of a laceration determines to a large extent the final outcome. He must learn to distinguish between those injuries amenable to repair in an Emergency Room and the more complicated problems requiring Operating Room facilities. The physician rendering the initial treatment for craniofacial trauma has indeed a great responsibility.

An evaluation of the entire patient must be made in order to establish priorities in management. This is particularly important when dealing with the patient with multiple wounds. An adequate airway must always be given first consideration; not only should it be established, but it must be maintained. Hemorrhage has to be controlled and shock corrected. Reduction of facial fractures may be postponed for several days (up to 2 weeks) until the patient's general condition will permit surgical correction.

When extensive lacerations are present, one must assume significant blood loss at the time of injury and during transportation to the hospital. Good management requires accurate assessment, blood cross-matching, preoperative intravenous therapy, and surgical repair in the operating room, where proper sterile conditions are present and the needed help is available. It is poor judgment, and unfair to the patient, to attempt extensive tissue repair when conditions are less than optimal. While we will concentrate on facial injuries and their repair, the techniques advocated are appropriate to any site on the body.

The face is the center of attention in social interaction, the dominant part of the body image, and the part most revealing of the personality traits of the person. Even after the physical scars have diminished, the

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psychological imprint of facial injury may leave its mark on the young man or woman for a lifetime. Physical and psychological rehabilitation of the facially disfigured patient is a long, tedious, and difficult process.[1]

TIMING OF REPAIR

Facial wounds should be closed as soon as possible within the bounds of good judgment. Lacerations involving deep soft tissue damage in the face should receive primary repair within the first 24 hours of injury. This appears to be a safe period because of the excellent blood supply of the facial tissues and the effectiveness of broad spectrum antibiotics if indicated. After this period, one should consider packing the wound open, and carrying out secondary repair 4 to 8 days later. The usual early use of tetanus toxoid or tetanus antitoxin following skin testing is still important.

WOUND PREPARATION

Prior to closure, soft tissue wounds must be properly prepared. They are first cleansed well under surgical conditions by washing thoroughly with white soap and water, using sterile cotton—never gauze. Gauze is rough and may abrade the skin. Except where frank dirt is impregnated in the wounds, cleansing is confined to the skin surface, and copious warm saline irrigation prepares the wound for closure. The addition of hydrogen peroxide does produce frothing and brings to the mouth of the wound loose particles and other debris. Antiseptics containing alcohol, iodine, etc., serve only to cauterize the healthy wound borders and invite inflammatory reaction in the wound; furthermore, *they hurt*! Aqueous zephiran solution (1:5000) is an adequate skin antiseptic and will not burn the eyes or exposed tissues.

Adequate anesthesia must always be provided. Local anesthesia using 0.5 to 1 per cent xylocaine with epinephrine, 1:100,000, is effective and preferable in adults and most children. In dealing with extensive wounds in a patient with airway problems, or with an uncooperative patient (particularly the very young child), general endotracheal anesthesia is preferable. Hair should be shaved around the wound to insure proper cleansing and ease of application and future removal of adhesive tape dressing. It is imperative that eyebrows be preserved since they provide accurate anatomic landmarks around which the edges of wounds may be approximated.

The greatest deterrent to wound infection is a healthy, viable wound produced through cleansing, atraumatic and meticulous handling of the tissues, and obliteration of all potential spaces by accurate approximation of all layers of tissue.

INGROUND FOREIGN MATERIAL

After being cleansed, the wound is inspected carefully, with a magnifying lens if necessary. Strict attention is paid to the presence of any

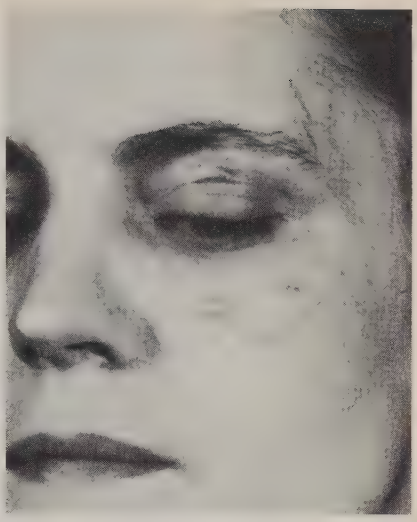

Figure 1. An "accidental tattoo" attributable to inadequate removal of inground street dirt at the time of the initial repair.

foreign material, which must be removed at once before it becomes "fixed" into the tissue edges of lacerations or into abrasions. Failure to remove it will leave permanent pigmentation in the skin, commonly referred to as an "accidental tattoo" (Fig. 1). If the material is grease or paint, it can ordinarily be dissolved with ether. If the foreign material is street dirt or gun powder, the area is scrubbed vigorously with a stiff brush. A No. 11 knife blade or a small curet may also be used. If the material is deep or difficult to dislodge, the use of a dermabrader or motor driven wire brush may be helpful. In case the discoloration is limited to the wound edges only, it may be better to debride these areas with a straight-edged scalpel. Anesthesia of the area to be cleansed or abraded is essential.

LACERATED WOUNDS

One must inspect carefully the edges of all lacerated wounds for evidence of contusion or small jagged edges that might become necrotic. Any segments that are doubtful of survival should be excised (Fig. 2). Most lacerations are tangential cuts and in almost all areas of the face their margins can be trimmed at right angles to the plane of the skin, extending out to viable, bleeding normal tissue. An important point in debridement of a tangential laceration is the excision of dermal and subcutaneous tissue at the wound margin, permitting a coaptation of the tissues with as wide a dermis to dermis contact as possible.

Atraumatic handling of these tissues with fine delicate instruments will minimize the chances of poor results. Rough handling of tissues leaves a wound which is an ideal culture medium for postoperative infections.

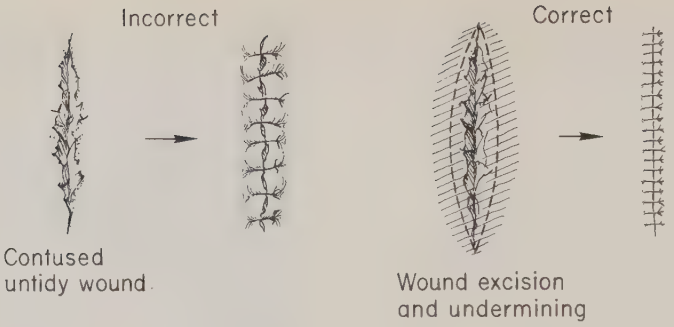

Figure 2. Skin laceration.

Complete hemostasis has to be achieved and the wound closed in layers without tension. This may necessitate undermining of skin and subcutaneous tissues adjacent to the wound edges. In initial control of hemorrhage, care is taken to avoid injurious vessel clamping, particularly over the distribution of the seventh cranial nerve. Crushing forceps and hemostats should never be used on the edges of pedicle flaps, as they leave areas of devitalized skin. Atraumatic skin hooks should be used to hold the edges of the skin flaps during the operative dissection.

A careful wound closure facilitates uneventful, primary healing. In order to achieve a satisfactory scar interrupted sutures of 5-0 and 6-0 monofilament nylon are used. They should be placed no further apart than 2.5 mm. and their point of entrance and exit on either side of the wound should be less than 2 mm. (Fig. 3). If the sutures are too tight, local pressure will lead to ischemia and necrosis, with subsequent scar formation and visible marks (railroad ties) (Fig. 4*A*). Also, tissue encompassed by snug sutures may become edematous, which indirectly in-

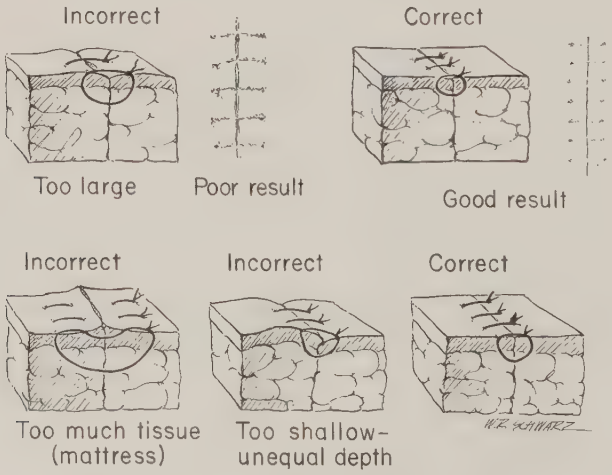

Figure 3. Suture technique.

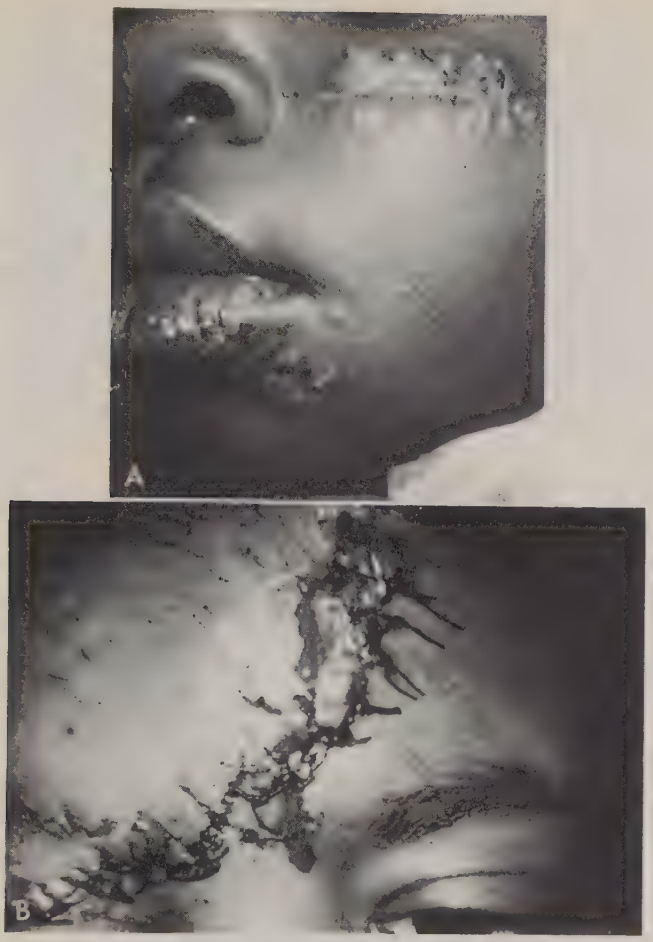

Figure 4. *A*, Permanent "stitch scars" resulting from intrinsic tension owing to tightness of the suture in relation to the encompassed tissue mass. *B*, Improper wound closure, overlapping of skin edges, sutures tied too tightly, large caliber suture material resulting in secondary wound infection.

creases tightness of the sutures.[2] Stitch abcesses may be caused by bacterial infection and are sometimes associated with necrosis caused by tight sutures (Fig. 4*B*). When the suture is removed, inflammation subsides but a permanent suture mark remains. Occasionally small horizontal mattress sutures, as necessary, may help to evert the edges.

The optimal time for the removal of skin sutures depends on the type and location of the wound, the accuracy with which the edges have been reapproximated, the gentleness with which the tissues have been handled surgically, and the thickness of the skin. Thin skin heals rapidly, so that eyelid sutures can be removed in 2 to 3 days. Thicker skin over heavy musculature is slow to heal and suture removal should await a longer period of time. Following suture removal support of the wound with strips of transparent adhesive for approximately three weeks will minimize separation of the wound edges (Fig. 5).

Figure 5. Traumatic laceration of cheek and chin repaired by cited principles and leading to an excellent result in a highly "kinetic area."

AVULSION INJURY

Avulsion injuries vary from a simple abrasion of the dermis to massive losses of skin, subcutaneous tissues, muscle, nerves, tendon, and bone. The success or failure of treatment is often determined in the first few hours after injury. For this reason, the surgeon treating an avulsion injury should have a clear and orderly plan of action.

If a large segment of skin has been avulsed, it should always be looked for at the scene of the accident. Unless it is hopelessly contused or mangled, it can usually be saved and utilized for repair, with a better final result than by substituting a skin graft from another site. Even though

grossly dirty, it can be prepared in the same way the wound was cleansed, soaked in an antibiotic solution, defatted, and reapplied to its original bed in the form of a free full-thickness skin graft.

Avulsion of large organs such as an entire nose or ear are serious catastrophies. Because of their large mass, they cannot be expected to grow if reapplied (Fig. 6). In total or partial avulsion of the ear, salvage of the underlying avulsed cartilage by "banking" it beneath subcutaneous tissues is advocated.

Human bites of the face are almost always true bites associated with loss of soft tissue or cartilage or both. The presence of the human oral organisms precludes reattachment of the avulsed segment. In general the patient is almost never seen early after a biting incident, particularly when the bite involves the face or neck. When the human bite wound is seen early, surgical debridement and primary suture or skin graft closure is advocated, provided that adequate systemic antibiotic coverage is maintained.[3] On the other hand, animal bites with puncture wounds should not be irrigated or closed. If the bite is seen relatively early and extensive tissue tearing is present, primary repair after debridement is indicated.

Skin flaps that have been partially avulsed are always precarious in their behavior. Invariably they have a better arterial supply than they do a venous return. This predisposes to venous congestion and moist gangrene. Flaps of this type can frequently be saved by deliberately dividing the remaining pedicle, removing the underlying attached subcutaneous fat, and then reapplying the skin as a free full-thickness graft.

The very common slice wound with beveled edges will often heal with contraction along the oblique tract of fibrous tissue, resulting in a

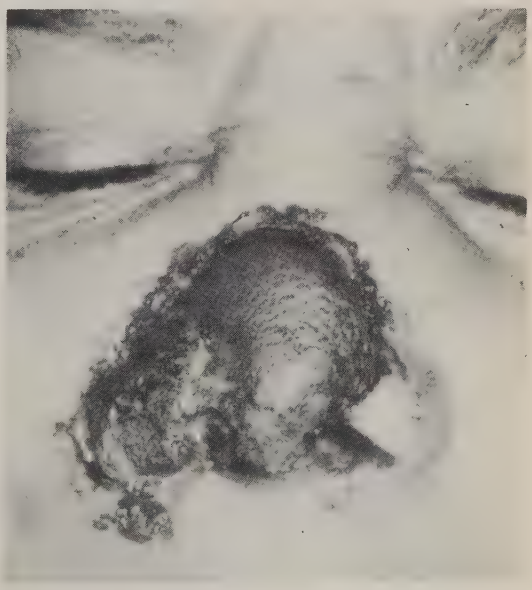

Figure 6. The reapplication of "composite" avulsed tissue consisting of skin, subcutaneous tissue, cartilage, and mucosa doomed to failure.

Figure 7. A typical partially avulsed retrograde semicircular flap of skin and subcutaneous tissue resulting in an edematous "trap door" deformity.

rolled or pouting wound margin. This is even more apparent in the "trap door" wound where the horseshoe-shaped scar also contracts, causing further pouting of the wound margin (Fig. 7). There is no completely satisfactory way of handling the initial injury. It is better to excise completely partial-thickness tags of skin that have been elevated rather than laying them back in place. The wound edges of the beveled cut may be excised so that the skin wound margins are vertical (Fig. 8). A firm pressure dressing is helpful and hematoma or seroma must be prevented.[4] In the first few months following injury, the soft tissue enclosed within the semicircular portions of the scar will thicken and lump up (pin cushion defect). After this period, there will be progressive softening and flattening as the scar becomes more pliable and elastic, so that spontaneous im-

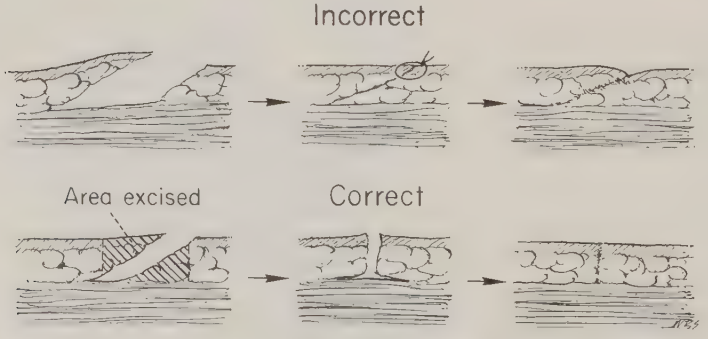

Figure 8. Repair of slicing wound. Suture materials: muscle, 4-0 chromic catgut; subcuticular ligatures, 4-0 plain catgut; skin, 5-0 or 6-0 nylon (silk).

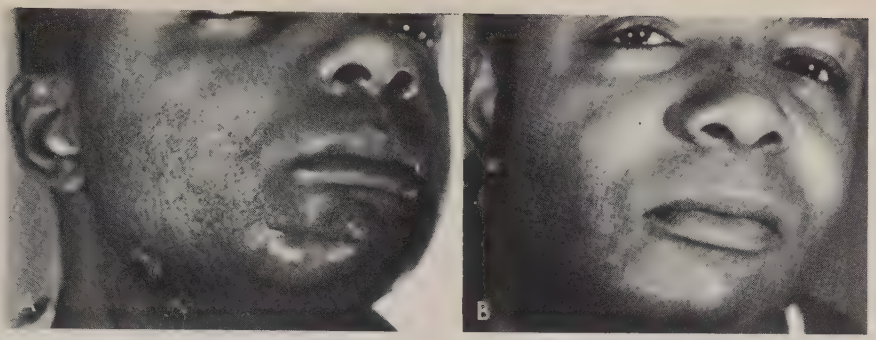

Figure 9. *A*, Horseshoe-shaped edematous partially avulsed flap on chin. *B*, Horseshoe shaped edematous partially avulsed flap following secondary plastic surgical revision one year later.

provement may be expected. Secondary repair usually involves excision of the scar and closure with the interdigitation of flaps (Z-plasty), so as to break up the circular direction of the wound and interpose normal elastic skin between segments of the scar (Fig. 9). If the flap was traumatically destined to be retrograde, then a chronic lymph edema may persist forever.

DEEP WOUNDS

All deep lacerations must be inspected carefully. Divided muscles should be reapproximated with interrupted sutures of 4-0 chromic catgut (Fig. 10). When muscle is severed, a two-layered closure is important, otherwise the muscle will retract, with hematoma filling the gap and eventually organizing to produce contracted scar tissue, and hence a depressed scar.

Injury to the parotid gland itself may result in a temporary external fistula; however, this type of wound generally responds to conservative management. On the other hand, an unattended injury to the main paro-

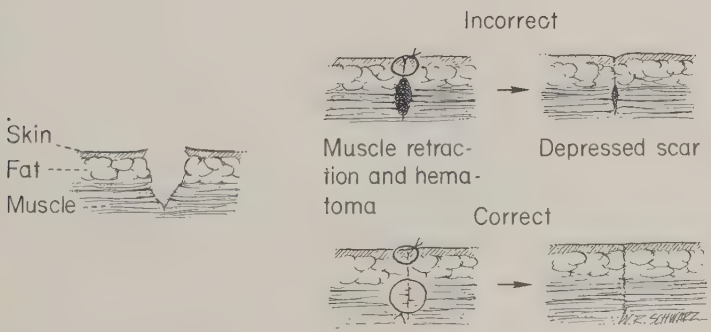

Figure 10. Repair of muscle injuries.

tid duct or one of its major branches inevitably results in a salivary fistula that is usually intractable to conservative therapy. The divided ends of the parotid duct must therefore be identified and repaired effectively using interrupted 6-0 black silk (Fig. 11). A thin polyethylene tube should be threaded through the repaired parotid or lacrimal duct and allowed to remain in place for 4 to 6 weeks. If only the proximal portion of the duct can be located, the surgeon can ligate it, thereby causing atrophy of the parotid gland.

Bisection of the main trunk or any branch of the facial nerve should be recognized prior to the injection of a local anesthetic. Immediate repair with interrupted 6-0 or 7-0 black silk in the perineurium is advocated. Lateral tarsorrhaphy may be advisable to prevent corneal exposure during the early postoperative period.

Surgical reattachment of severed palpebral ligaments also requires primary repair. They are rarely seen without concomitant fractures. The most important point in the repair is to place the suture deep enough back in the orbit. Most errors in repair are from suturing too superficially, leaving the injured canthal area positioned too far forward thereby not allowing the puncta to be in contact with the globe (Fig. 12).

When confronted with through-and-through lacerations of the lips or cheeks, surgical closure must be extremely accurate, particularly at the vermilion border (Fig. 13). In anticipation of considerable edema, it is best to place only a few strategic absorbable sutures in the mucosal layers. Through-and-through lacerations of the tongue should be primarily closed; even a portion of the tongue hanging by a small pedicle should be repaired since the tongue has such an excellent blood supply and amazing powers of survival.

Repair of eyelid lacerations is best carried out in the operating room where proper instruments, suture material and assistance are available. Great care should be taken to approximate the tarsus and ciliary margin accurately (Fig. 14). After the structures have been sutured, the remainder of the eyelid generally falls into place. Z-plasty procedures at the lid margins may be necessary.

DRESSINGS

The management of the traumatized area following satisfactory closure contributes considerably to an eventually successful result. The initial dressing should consist of nylon fabric immediately over the sutured or abraded wounds plus a bulky dressing with adhesive qualities supporting the tissues in the desired position. These will maintain the tissues in close approximation thereby diminishing "dead space" and arrest tendency of hematoma formation and minimize tension on the suture line. If indicated, suction drains should be removed in 48 to 72 hours and care must be exercised in their proper placement in order to prevent pressure necrosis of the overlying freshly traumatized skin. After 2 or 3 days the value of such dressings is questionable. Meticulous care to prevent crusting by the wiping of the wound edges with cotton tip applicators

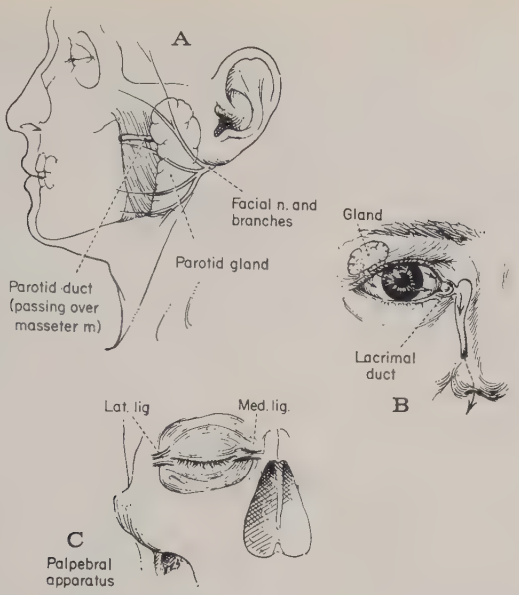

Figure 11. Structures requiring primary repair.

Figure 12. *A*, Markedly displaced medial canthus of right eye owing to improper stabilization of palpebral ligament at time of primary repair. *B*, Result after secondary repair with reattachment of medial canthal ligament (through and through wire suture to nasal bones) and dacryocystorhinostomy.

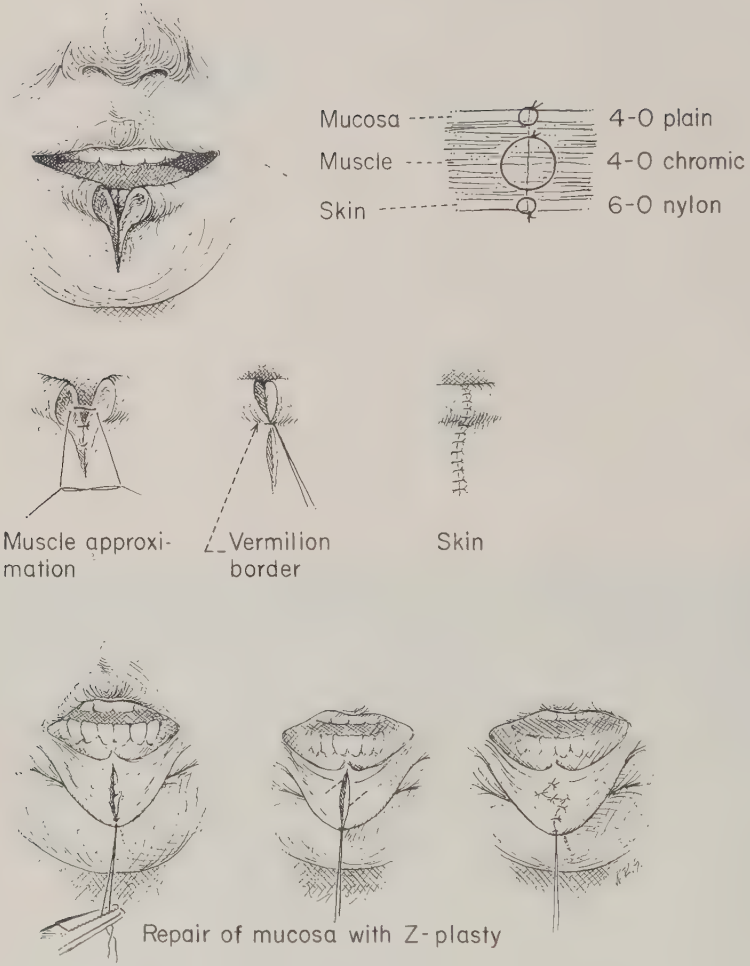

Figure 13. Repair of through-and-through lip laceration.

Figure 14. Ragged partially avulsed laceration of upper and lower eyelids repaired using basic plastic surgical principles.

soaked in hydrogen peroxide is our personal recommendation to the patient. Following the removal of all sutures, support of the wound by strips of tape will prevent wound spread. Micropore surgical adhesive tape or clear plastic tape should be applied perpendicular to the wound edge. It should be removed daily for face cleansing and reapplied, for a period of at least 3 weeks.

SUMMARY

In the treatment of craniofacial trauma an evaluation of the *entire* patient must be made in order to establish proper priority of management.

An adequate airway must be established, shock corrected, and a condition of adequate anesthesia (local or general) achieved before cleansing, inspection and repair of facial lacerations.

The greatest deterrent to wound infection is a healthy viable wound produced through cleansing, atraumatic and meticulous handling of the

tissues, and obliteration of all potential spaces by accurate approximation of all layers of tissue.

Early surgical closure is emphasized. Descriptions of management of superficial, deep, and compound lacerations and avulsions as well as enumeration of all important deep structures of the face encountered are included.

A good final result in this most important anatomical area can be promised by attention to detail in a region in which a rich blood supply is our greatest ally.

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The Management of Head Trauma

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The head is one of the most vulnerable organs to trauma, and man has long recognized the serious consequences of this type of injury and attempted to prevent and treat it. This can be deduced from the helmets so frequently observed in ancient Persian, Greek, and Roman sculpture, and by the fact that trephinations were performed on skull fractures even in the prehistoric era.[6]

In spite of the long recognition of the importance of head injury, however, relatively little has been done to protect the brain effectively against trauma, and little is known about the treatment of severe brain injuries. Today head injury is the highest cause of death in patients between the ages of 1 and 44 years, and is the cause of death in two thirds of all fatal accidents.[9] Traffic accidents are the highest cause of trauma in the United States.[1] Multiple injuries are more frequent than single injuries in these accidents, occurring in about 70 per cent of all injured persons. The head is involved in 60 to 70 per cent of all traffic accidents[7, 9] and is the most frequent cause of death.

If we divide the body into six areas – head, neck, thorax, abdomen, upper extremities, and lower extremities – the high risk of mortality from trauma increases from an average of about 6 per cent for one area to about 60 per cent for six areas. It is important to remember that the head and neck increase the risk disproportionately as compared with other areas. That is, while on the average the high risk for two areas of the body is about 10 per cent, the risk is 31.6 per cent when head and neck are involved, in contrast to 0.1 per cent for upper and lower extremities.[7]

PRIORITIES IN EVALUATION AND MANAGEMENT

The priorities in evaluation and management of patients with head injury are as follows: respiration, circulation, and the nervous system.

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Respiration

Patients with severe head injury frequently develop respiratory difficulties. Our clinical experience with patients and laboratory experiments with animals clearly show that any labored respiration is associated with a rise in intracranial pressure. The rise of intracranial pressure is abrupt and significant when there is airway obstruction, marked slowness of the respiratory rate, and periods of apnea. The rise of intracranial pressure in airway obstruction most likely results from high venous pressure which hinders the return of the blood from the cranial cavity. On the other hand, the respiratory difficulty itself is frequently in part or totally a result of brain stem injury or increased intracranial pressure. Increased intracranial pressure and respiratory difficulties affect one another adversely, producing a vicious cycle. It is, therefore, highly important to determine the cause of the respiratory difficulty and to correct it immediately.

The respiratory problems in head injuries commonly result from upper airway obstructions. In upper airway obstruction the respiration is noisy and prolonged, and sometimes abortive, both with inspiration and expiration. The obstruction is usually caused by the root of the tongue falling backward in comatose patients, nasopharyngeal secretions, vomitus, blood, and cerebrospinal fluid from basal skull fractures. At times the obstruction can be relieved simply by holding the chin upward, using an oralpharyngeal tube to hold the tongue up, and/or aspirating of the nasopharyngeal region. If the obstruction is not relieved satisfactorily by these simple means, tracheal intubation or tracheostomy is required.

Tracheal intubation is preferred to tracheostomy, because it can be done easily and rapidly, serves an often temporarily needed purpose, and facilitates a tracheostomy if it proves to be necessary later. Tracheal intubation may be difficult in patients who are semiconscious and agitated. Tracheostomy is tolerated better in that it causes less cough and discomfort, and secretions can be cleared more efficiently. Tracheostomy is preferred when cervical spine or facial bone fractures are suspected.

Circulation

Shock does not usually develop in an individual with intracranial bleeding unless he has reached a terminal stage. Therefore, shock in a patient with head injury suggests an extracranial cause. The shock may be hypovolemic, normovolemic, or neurogenic. A patient with mild head injury might appear worse when there is associated shock, and one might make erroneous decisions for unnecessary neurosurgical intervention for the following reasons:

1. Restlessness, confusion, generalized weakness, fainting, or bilaterally dilated pupils could all result from shock and be confused with the signs of increased intracranial pressure.

2. Shock in the presence of cyanosis and high venous pressure which is commonly of cardio-pulmonary origin, such as that seen with cardiac tamponade, collapsed lung, pneumothorax, hemothorax, and fractured ribs, is associated with high carbon dioxide retention and cerebrovascular

dilation, giving different degrees of suppression of the level of consciousness.

3. Head injuries, especially in traffic accidents, may be associated with cervical spine injury (10 per cent).[3] Besides the paralysis and paresis which occur in spinal cord injury, the cervical cord compression might produce shock or decreased respiratory capacity and alter consciousness.

Nervous System

Having ruled out the need for or having accomplished the primary support of respiration and circulation, the evaluation and management of the head injury begin. The level of consciousness, the size of the pupils, and the presence or absence of paralysis and vital signs are the first and the most important aspects to be evaluated.

Level of Consciousness. The level of consciousness is perhaps the most reliable sign by which to judge the extent of cerebral injury. Except for depressed skull fracture or penetration of the brain by sharp objects, the chances are great that, no matter what other abnormal neurologic signs are present, if consciousness is normal the patient is in no need for immediate cranial neurosurgical intervention. It is when the level of consciousness is disturbed that the examiner's knowledge, experience, and the "art of medicine" suddenly begin to play a vital role.

Pupil Size and Response. The second most reliable sign in the head-injured patient is alteration in pupil size and response to light. The pupillary response to light becomes slower, and the size becomes larger as cerebral compression increases. This type of alteration is frequently unilateral at first, and nearly 90 per cent ipsilateral with the clot, becoming bilateral later. In sudden and marked increased intracranial pressure, both pupils dilate rapidly and simultaneously.

Paralysis. Different degrees of paralysis and paresis can be seen following head injury. While hemiparesis may be associated with an intracranial clot, the hemiparesis might be concealed by coma. An intracranial clot large enough to give signs of hemiparesis would certainly disturb the consciousness. Therefore, with the exception of depressed skull fracture, when there are different degrees of local paralysis or paraparesis in a conscious patient, spinal cord and peripheral nerve injury should be kept in mind. Similarly, fractures or dislocations might at times erroneously be interpreted as paresis.

Alterations of Respiration, Pulse, and Blood Pressure. Alterations in respiration, pulse, and blood pressure—the so-called vital signs—are important signs in increased intracranial pressure. Using an extradural bag filled with mercury, Cushing[2] produced cerebral compression in animals, and found that as intracranial pressure was raised, respiration and pulse decreased but the blood pressure increased. This phenomenon, the Cushing triad, has long been observed as an important factor for evaluation and observation of patients following head injury.

Our laboratory studies reveal[4] that as intracranial pressure is increased in cats through an extradural balloon, the respiration decreases in the earlier stages of intracranial pressure, and is followed by slowness of pulse (Fig. 1). The arterial pressure rises in the later stage when the

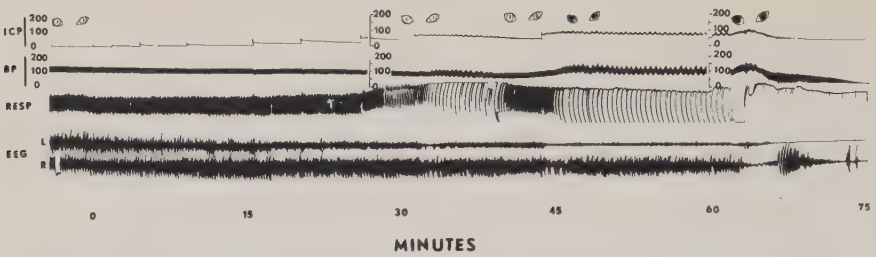

Figure 1. The alteration of EEG, respiration (Resp), pupils and blood pressure (BP) as the intracranial pressure (ICP) is raised by gradual inflation of an extradural balloon in a cat. The recording paper is run too slowly here to analyze the details of the EEG, but note that the amplitude of the EEG is decreased in the early stage and decreased first on the left side (L), the side of cerebral compression. The respiration and pulse (not shown here) alter before the ipsilateral pupil dilates and blood pressure rises later when both pupils are dilated. (*From* Hekmatpanah, J.: Neurosurg., 32:16–20, 1970, by permission.)

intracranial pressure is markedly high. While the pulse frequently decreases with the rising intracranial pressure, it may become fast when the arterial pressure is rising. Similarly in patients with head injury, the rise of the blood pressure is a late sign and cannot be depended upon as a warning sign for early rise of intracranial pressure.

In acute head injury and especially when there are multiple injuries, the respiration and pulse may be altered by mechanisms other than intracranial pressure. However, slowing of the pulse is often a reliable sign of increased intracranial pressure.

TYPES OF INJURY

The conditions of respiration, circulation, and the central nervous system can usually be evaluated within the first few minutes of examination, and the priority for management be planned. If the above mentioned signs are normal, an angiogram is seldom needed initially unless the patient's condition changes following observation. In trauma to the head, there may be one or a combination of the following conditions.

Lacerations of the Scalp

While the detailed explanation of the management of different scalp lacerations and avulsions are avoided in the interest of brevity, the following should be considered.

1. The scalp laceration should be managed as quickly as possible, since even a small laceration is apt to give serious complications for two reasons: (a) the scalp is very vascular and can bleed profusely, causing unnecessary need for transfusion; and (b) laceration of the scalp, particularly when associated with skull fractures, can cause meningitis or brain abscess, which are serious and unnecessary complications.

2. Shaving a wide area around the wound, irrigation, removing of foreign bodies, and debridement (if needed), which are necessary procedures to prevent infection, may be associated with continuous

bleeding. Therefore, speed and especially the presence of an assistant to compress the edges of the wound against the skull can be of considerable value in preventing blood loss.

3. Unless a large vessel such as the temporal or occipital artery is torn, there is no need for tying the individual bleeding arteries. It is usually a futile and time-wasting process.

4. Not only may a two-layer closure of the scalp laceration be unnecessary; it can also promote infection. Even when the galea is torn, a fast closure of the scalp with a needle large enough to go through the whole thickness with good approximation is all that is needed to stop the bleeding and ensure healing.

5. Large or multiple lacerations, avulsions, or lacerations associated with depressed skull fractures require more specialized care in an operating room, where better facilities for care are available.

6. If a patient with an open depressed skull fracture has to be transferred to another hospital, a temporary closure of the scalp to prevent hemorrhage and infection may be necessary.

Skull Fractures

LINEAR SKULL FRACTURES. Linear skull fractures are usually caused by low velocity impact, in which the skull is struck against a large surface area. The linear fracture over the convexity of the skull is frequently without any specific sign and is depicted on skull films. Sometimes, especially in children, the fracture is associated with a tear in the dura, causing the collection of cerebrospinal fluid under the scalp, and is confused with subgaleal hematoma.

The fractures running through the base of the skull might be associated with bloody cerebrospinal fluid through the nose (rhinorrhea), pharynx, or ears (otorrhea). In fractures of the base of the skull a bluish discoloration around the mastoids might appear a day or two later (Battle's sign). Sometimes the blood in the external auditory canal is caused by a local injury in the canal or from outside contamination, and is not the result of basal skull fracture.

Linear skull fractures do not usually need any specific treatment. However, if the skin over the fracture is open or if the fracture at the base of the skull goes through the paranasal or mastoid sinuses, antibiotics (usually penicillin) are administered for several weeks. Except for occasional instances, most traumatic cerebrospinal fluid leaks stop spontaneously within 2 to 4 weeks. Therefore, operative repair of the dural tear, which used to be considered so strongly before the antibiotic era, is not, in our opinion, indicated unless the leakage continues beyond 4 to 6 weeks after the trauma.

DEPRESSED SKULL FRACTURE. Depressed skull fracture is commonly caused by high velocity impact or a strike over a small surface area of the head. A depressed skull fracture might cause different degrees of focal cerebral contusion, manifested by disturbance of speech or local neurological signs. It may tear the dura or venous sinuses. Practically all depressed skull fractures require elevation. While opinions differ as to the urgency of the need for elevation with intact overlying scalp, there is common agreement about the need for immediate

operation for an open fracture, to prevent meningitis. A depressed skull fracture might tear or obstruct cerebral venous sinuses. For this reason we prefer to elevate all depressed skull fractures immediately if at all possible. When the fracture overlies the sagittal or lateral sinus, a bone fragment might enter the sinus and cause profuse bleeding when it is removed. For these cases it is wise to be prepared for the possible need of blood transfusions and to have adequate exposure before removing the fragment.

Concussion

An ad hoc committee to study head injury nomenclature[8] first met in Chicago in 1964 and defined concussion as "A clinical syndrome characterized by immediate and transient impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium, etc. due to mechanical forces." The pathophysiology of concussion is, however, not very well known. Theories such as neuronal excitation, nerve cell injury and death, and cerebral ischemias have been advanced. In concussion, the duration of unconsciousness following trauma is usually minutes (Fig. 2). Commonly patients with transient loss of consciousness following injury show little or no evidence of neurologic or mental disabilities later.

Epidural Hematoma

Epidural hematomas are commonly caused by a tear through the meningeal arteries and are usually located in the temporal region. About 90 per cent of the epidural hematomas are associated with skull fracture. Except for occasional cases, the fracture is located on the side of the hematoma. In this condition, the patient might lose consciousness for several minutes, owing to concussion following the trauma, but the level of consciousness decreases again and the patient becomes comatose over a period of several hours (Fig. 2). The duration of time between the first and second periods of unconsciousness is called the lucid interval. The lucid interval may be as long as a day or two in occasional cases.

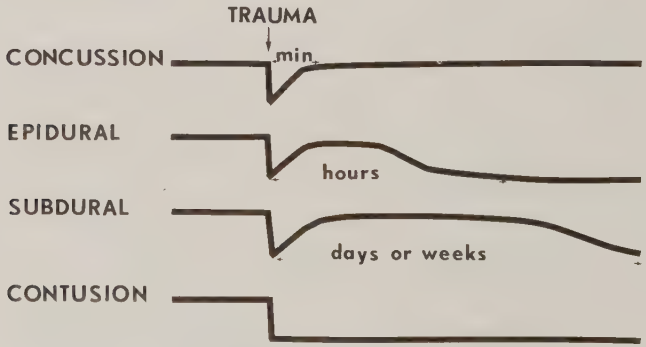

Figure 2. Alteration of consciousness level after trauma. Note the initial and transient loss of consciousness (concussion) for several minutes and decline of consciousness within hours in epidural hematoma, and within days or weeks in subdural hematoma. In severe contusion the patient might remain permanently unconscious.

Subdural Hematoma

Subdural hematoma is caused by venous bleeding beneath the dura following head injury. Bleeding usually occurs from a torn bridging vein between the cortex and the dura. Subdural hematoma may occur with very minor trauma in patients taking anticoagulants, or spontaneously in association with ruptured aneurysms or arteriovenous malformations. Classically, subdural hematomas are divided into acute, subacute, and chronic types. While the bleeding occurs at the time of the trauma, the symptoms and signs of cerebral compression may appear at different periods of time afterward. The symptoms of acute subdural hematoma occur right after head injury, and those of chronic hematoma occur several weeks later.

An acute subdural hematoma results from massive and frequently bilateral bleeding, commonly from torn dural sinuses. Usually the patient's condition is serious and deteriorates rapidly. The mortality of acute subdural hematoma is around 90 per cent. Immediate trephination of the skull and removal of the hematoma may be life-saving.

In chronic subdural hematoma the lucid interval is several weeks (Fig. 2) and sometimes a few months. During this time, the most common findings are headache and confusion. Finally the patient becomes drowsy, comatose, and eventually dies if not operated upon. Because of the paucity of symptoms and signs in chronic subdural hematoma, the diagnosis could easily be missed if not suspected (Fig. 3), especially when there is mild fever and leukocytosis owing to dehydration or superimposed pneumonitis.

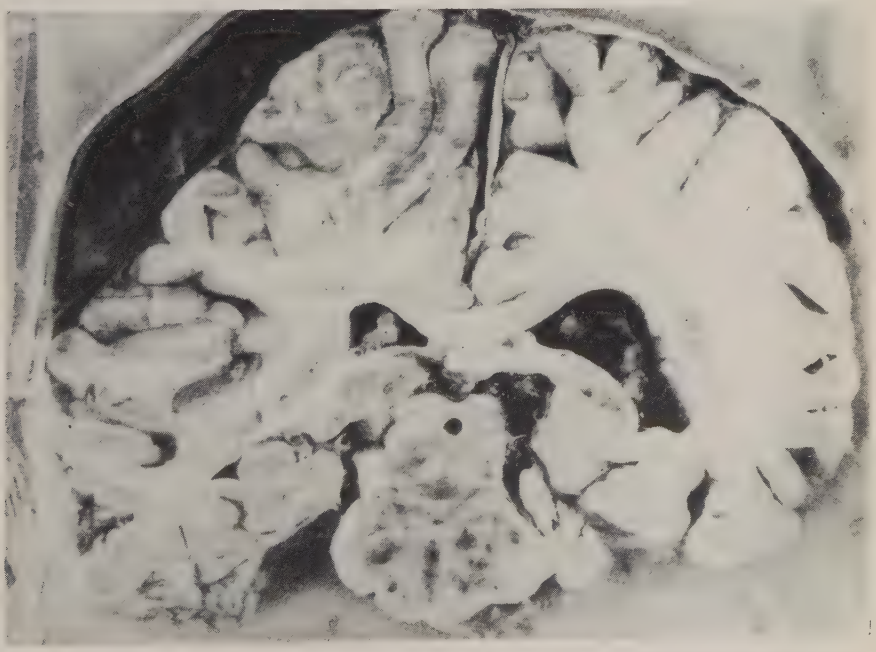

Figure 3. Subdural hematoma in an elderly patient with a recent history of headaches, confusion, and mild fever, who has died without the benefit of diagnosis and operation.

Chronic subdural hematoma is more commonly seen in elderly or alcoholic patients. In these patients the subdural space is large and the hematoma may exist without any clinical evidence of increased intracranial pressure. Papilledema is occasionally noticed, but is often absent. A normal cerebrospinal fluid pressure is frequently found in elderly patients with subdural hematoma. A misleading symptom is fluctuation in the level of consciousness; the patient who is drowsy on admission wakes up later and is discharged, only to return later in deeper coma.

At times, patients with subdural hematoma make repeated visits to the emergency room, without being admitted. This is unfortunate, for the chronic subdural hematoma is usually curable, if the clot is removed before the patient becomes deeply comatose. Therefore, particularly in elderly patients with a recent history of headaches and confusion, subdural hematoma must be suspected and ruled out. The patient or his family may not know of or remember the trauma. When any one of the following findings is also present, the index of suspicion is high and attempts for evaluation should be made by angiography or burr holes: (1) a history of fall several weeks previously; (2) ecchymotic area on the face, head, or any other part of the body; (3) slow respiration and slow pulse; (4) a recent change in sleeping habits, with longer sleep in the mornings; (5) a recent change in memory, orientation, and ability to continue previous functions and duties; and (6) a linear skull fracture on skull films, or shift of the pineal gland.

Brain scanning is positive only in about 50 to 70 per cent of patients with chronic subdural hematomas. Echoencephalography is frequently helpful, but may fail to show a midline shift. If the index of suspicion is high, angiography should be carried out, in spite of normal brain scan and echoencephalography. Angiography is most helpful for evaluation of subdural hematoma; an avascular space is seen between the cortical vessels and the calvarium on the anteroposterior view (Fig. 4). A lateral view angiogram is necessary to rule out a frontal subdural hematoma, as this might be missed on the anteroposterior view.

The treatment of subdural and epidural hematomas is surgical. Occasionally a thin subdural hematoma is absorbed spontaneously. A recent subdural hematoma takes the form of a solid clot and cannot be removed through burr holes. Subdural hematoma of several weeks' duration is usually liquified and can be removed through burr holes. For this reason, three burr holes are made so that they can be connected and turned into a craniotomy flap if the clot is solid (Fig. 5).

Although angiography is highly important and frequently done for the evaluation of subdural hematoma, at times it is a time-wasting process. In a patient whose condition is deteriorating, it is far preferable to perform trephination immediately without waiting for angiographic diagnosis.

Not infrequently, subdural hematoma is bilateral. This should be suspected if a positive angiogram shows no midline shift. Also, subdural hematoma may be present without midline shift. For this reason, if the suspicion is high, an angiogram on the opposite side is indicated if the

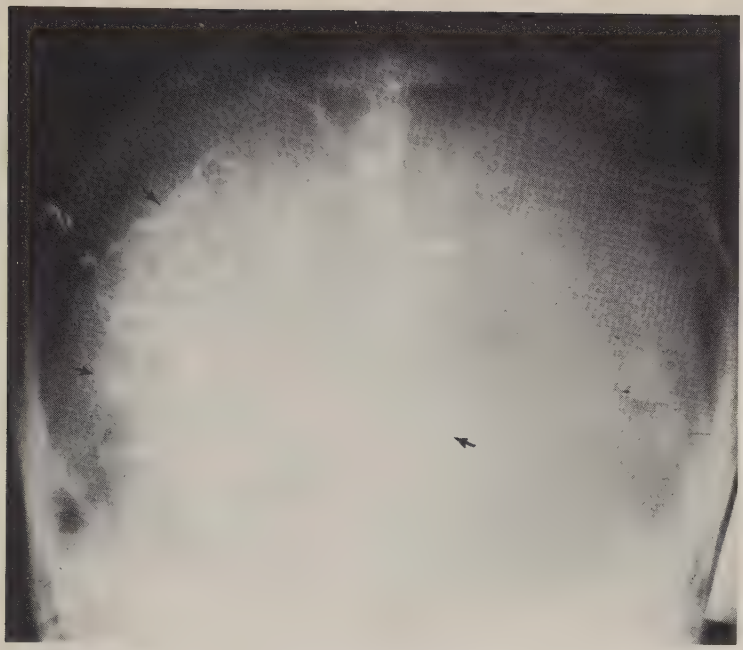

Figure 4. Subdural hematoma (upper arrows) in a 70 year old patient with a 10 week history of headaches, confusion, and dizziness, until he became comatose. Note the shift of anterior cerebral. The pineal (central arrow), now shifted, was still in the midline 2 weeks after trauma.

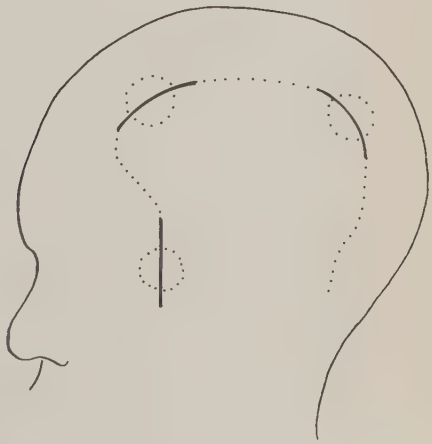

Figure 5. The position of incisions (solid lines) for burr holes (dotted circles). The incisions can be connected and extended to a flap if the clot is solid.

first side is normal. Angiogram of the opposite side is seldom needed, however, if the first side shows a subdural hematoma, since burr holes are commonly made on both sides.

Cerebral Contusion

Cerebral contusion may be local, in which case one finds a local neurologic abnormality with or without disturbance of consciousness. Diffuse and generalized cerebral contusion is usually associated with immediate and long-standing coma. The morbidity and mortality is usually around 90 per cent and in those patients who do recover, different degrees of physical and mental disability remain. A local or generalized cerebral contusion may be confused with a subdural or epidural hematoma, but angiography will help to rule out hematoma.

DISCUSSION AND CONCLUSION

Trauma, like pollution and war, has become one of the major man-made sources of public health problems. It is astonishing to think how man's ineptitude has allowed such catastrophes to develop and to creep into his life, and, like cancer, to erode and threaten his existence. And this is the very same existence that he has spent so much of his life and energy to protect.

About ten million disabling injuries, with over one hundred thousand deaths, occur yearly in the United States. Accidents cost the public twenty-five billion dollars each year. Motor vehicles are the main cause for serious injuries, with over fifty thousand deaths and at a cost of twelve billion dollars.[1] Head injury ranks high among injuries, and it occurs in about three million persons, causing death to about fifty thousand.

Without attempting to pretend that all the influencing factors for this drastic and universal social neglect are known, the following may well be strong factors in the morbidity and mortality of head injuries.

1. Nerve cells are very sensitive to oxygen and glucose deprivation of more than several minutes. In head injury, one encounters increased intracranial pressure, and laboratory studies[5] indicate that when the intracranial pressure rises, the cerebral blood flow decreases. Cerebral blood flow slows from the very early stages of increased intracranial pressure. The flow is markedly slow when one of the pupils is dilated, and it is practically stopped when both pupils are dilated. In rising intracranial pressure and slowing circulation, the red cells sludge and form microemboli, which occlude the vessels from inside. The vessels are also compressed from outside pressure. This cessation of circulation in an increasing number of vessels causes an increasing number of deaths in cell population.

2. In severe head injury the intracranial pressure rises very fast, and the maximum injury occurs in a short time, before the patient reaches the hospital.

3. While the decision for management should be made very rapidly, if the brain is to be protected against damage, the patient is commonly

evaluated and treated first by those least expert. This is true for both civilian and military injuries. In most countries, about 90 per cent of head injuries are treated by general surgeons.[3] Medical students are as a rule inadequately trained to recognize neurologic disease, a shortcoming which is bound to influence the results of treatment.

4. Respiratory and circulatory problems influence the signs and symptoms of head injury to a great extent. These important factors might not be recognized and corrected, which surely increases the morbidity and mortality. Not infrequently, one encounters a patient with airway obstruction, shock, or both, who on first sight appears to need immediate craniotomy, but, following the correction of airway obstruction or shock, is found to have only a minor head injury, with no need for surgical intervention.

6. Head injury, like other injuries, occurs accidentally, and patients are brought to the hospital without appointments. One can presume that this unexpected irregularity has not captured the sympathy of a great number of physicians, and as a result relatively few have become experts in the management of injuries.

7. Finally, government and public support for research in head injury is disproportionately low as compared with support for other serious illnesses, such as cancer and heart disease.

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Craniocerebral Trauma in Children

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This paper is based upon 246 consecutive admissions of children who have sustained craniocerebral trauma to the Children's Memorial Hospital of Chicago and Loyola University Medical Center, Maywood, Illinois. Clinical aspects of various head injuries which occur in children will be considered. Injuries discussed include cephalohematoma, linear fractures at birth, both open and closed, depressed skull fractures, leptomeningeal cysts, hematomas of the intracranial compartment, and cerebral contusion. Special emphasis will be placed on the characteristics and treatment of chronic subdural hematomas, and the angiographic criteria for diagnosis.

CLINICAL ASPECTS OF HEAD INJURY IN CHILDREN

It is of great interest that loss of consciousness is related to the age of the child. The younger the child, the less likely it is that the child will lose consciousness, even though trauma is severe. Loss of consciousness, per se, does not necessarily correlate with the prognosis. Of our 246 patients only 20 children between 1 month and 2 years of age lost consciousness; 60 children between 2 and 4 were unconscious; 90 between 4 and 6 years were unconscious for a prolonged period of time. After regaining consciousness, the last group remained stuporous for a period ranging between 1 and 6 days.

Vomiting is a rather common symptom in the younger child. Children in the first year of life seldom have emesis, but from 2 to 12 years of age it was present in 85 per cent of our patients. The presence of the sign was not necessarily associated with the loss of consciousness. It is impossible, needless to say, to evaluate headaches in children around the age of 2. The presence of symptoms increases conspicuously with the age of the child, and headache could be a long-standing complaint even though the child is neurologically sound otherwise. Headache was present in 70 per cent of children between 2 and 14 years of age.

Convulsion may also occur following a head injury although it is not

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common. In our series 15 children between the ages of 8 months and 2 years had Jacksonian seizures and only 20 over 2 years of age had grand mal seizures. Only 2 children had status epilepticus; 11 children between the age of 2 and 8 years developed post ictal palsy in spite of the fact that the convulsion was of the grand mal variety. The only permanent neurologic deficit was seen in those patients with focal seizures, and in each instance the child had significant brain damage.

Involvement of the cranial nerves is a rather rare sign in infants and newborn babies. Of the entire group 34 patients had sustained perforation of the tympanic membrane with clinical evidence of basal skull fracture and cerebrospinal fluid otorrheas, but only 4 had peripheral seventh nerve palsy. In no instance was surgical intervention necessary to correct it.

Two children from 10 to 14 years of age had third nerve involvement. One had a cavernous sinus fistula with complete ophthalmoplegia in the right. In order of frequency the other cranial nerve most commonly involved was the sixth nerve; in 3 cases involvement was unilateral, and in 6, bilateral.

One rarely finds lateralizing signs in newborn infants. The brain injury in this age group is most commonly manifested by irritability, periods of lethargy, or changes in feeding habits. Of these 246 cases, 47 had hemiparesis and the ages ranged between 6 months and 14 years. In children with acute head injury one seldom sees the classic Cushing phenomenon described in the adult; on the contrary, the pulse is rapid and the blood pressure is low because the blood volume of the infant and newborn is very small, and a large portion of the circulating blood may be harbored in an epidural or subdural hematoma.

The pupil size as well as the reaction to light offers to the clinician invaluable help. Pin-point pupils in trauma speak in favor of a severe brain stem contusion or intraventricular hemorrhage. Anisochoria (unequal pupils) usually indicates compression of the third cranial nerve on the side of the dilated pupil; however, one should never forget that craniocerebral trauma most of the time is accompanied by traumas of the cervical spine and spinal cord.

Although only one case of Horner's syndrome was observed in this series, one should always be alert to the fact that craniovertebral trauma does occur, and for this reason every traumatized patient should be cautiously mobilized. Particular care should be taken when x-rays of the skull are ordered since many x-ray technicians turn the head of the patient to the side when lateral views of the cranium are taken. This, of course, could produce irreparable damage to the spinal cord. It should be emphasized that in our series, out of 140 surgical lesions, only 10 presented anisochoria; therefore, one should not rule out space-taking lesions because of the lack of pupillary changes.

Together with the pupillary evaluation, the eyeground examination is also of vital importance. One would not expect papilledema to be found at the moment of the injury, but retinal detachment should always be considered. The eyeground in acute intracranial pressure may show pools of retinal hemorrhages. The increase in pupillary size and the number of hemorrhages speak for aggravation of the patient's clinical condition and

constitute an indication for emergency cerebral arteriography. Out of 70 patients with cerebral contusions, 10 had retinal hemorrhages and 2 retinal detachments. Of 40 acute subdural hematomas 12 had pools of retinal hemorrhage. Retinal hemorrhage was present in 4 cases of 85 chronic subdural hematomas seen.

Cerebrospinal fluid otorrhea was present in 34 cases of basal skull fracture at the mid-fossa. Five of these patients had a concomitant surgical lesion, 2 had acute subdural hematomas, and 3 had surgical temporal lobe contusion. Rhinorrhea was rarely seen, and when present it was associated with otorrhea. It is possible that the cerebrospinal fluid was coming from the eustachian tube into the nasal cavity.

The spinal tap in the child with an acute head injury is of little value and may even be very dangerous since cerebral herniation may occur following the evacuation of fluid. Furthermore, the possible tamponade effect over a bleeding vessel may be lost, and the bleeding may, of course, become more active.

FRACTURES AT BIRTH AND LINEAR SKULL FRACTURES

There is unanimity in the literature that fractures at birth are rare phenomena: 3 have been observed, 2 parietal and 1 in the occipital region. This type of fracture requires no treatment. Rarely, encephalohematomas become calcified, and removal is indicated for cosmetic reasons.

Linear skull fractures were present in 70 patients in our series and if no underlying intracranial pathology is present surgical treatment is not necessary.

LEPTOMENINGEAL CYSTS

The leptomeningeal cyst, called a "growing skull fracture" in the child, was first recognized about 100 years ago as a complication of skull fractures, but received very little attention up until 1940. Since that time there have been several reports in the literature dealing with this condition. Krauseon in 1907 called this condition "circumscribed cystic arachnoiditis". Others called it hydrocephalus external circumscripta. Schwartz, in 1941, described the mechanism that leads to the evaluation of the effects. Taveras in 1953 reported in detail the signs and symptoms of 7 children who were operated on for leptomeningeal cysts.

Of our 246 patients only 4 had leptomeningeal cysts. In general, it is agreed that the bony skull fractures in children will heal spontaneously unless there has been damage to the underlying dura. Bone defects may appear from 4 months to many years after the fracture has occurred. (Figs. 1 and 2). When the dura below a fracture line is torn, and tissue, generally leptomeninges, herniates through the defect, normal apposition and healing does not occur. The pulsation of the growing brain and the cerebrospinal fluid transmitted by the circle of Willis and other arteries of the brain, located in the subarachnoid space, push the meninges through

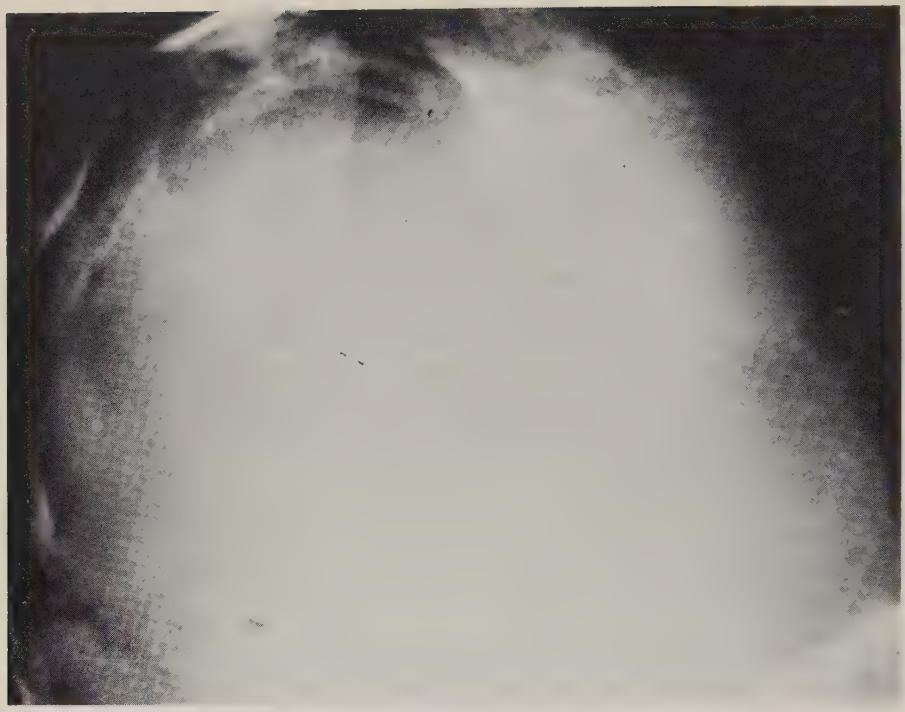

Figure 1. Growing skull fracture.

the tear. The encarcerated subarachnoid fluid, with the constant pulsatile pressure creates forces against the rims of the fracture. Consequently, bone absorption occurs with secondary enlargement of the previous linear fracture.

Headache and epilepsy are two symptoms of leptomeningeal cysts. Epileptogenic foci may occur with secondary brain damage, sometimes owing to occlusion of trapped arteries that supply the brain, causing cerebral infarction which can be confirmed on occasion angiographically (Fig. 3). The usual physical findings that should alert the physician are the soft bulging mass at the site of the fracture, about 2 months after a linear fracture has been found. It is possible to palpate the skull defect. Radiographic evidence of an increase in the size of a linear fracture is pathognomonic of this condition. The surgical correction of this entity should be accomplished by complete removal of the cyst, rather than simply invagination of the herniating portion. Cranioplasty should always be performed.

INTRACRANIAL HEMATOMAS

Acute Subdural Hematomas

An acute subdural hematoma is usually accompanied by a severe brain contusion. One common practice in the child with a head injury is

the subdural tap. Rapid evacuation of blood from the subdural space may be a life-saving procedure and should be done in four directions, from the outermost corner of the anterior fontanelle, away from the midline, anteriorly, posteriorly, laterally, and medially. A properly done, positive subdural tap confirms the presence of a subdural hematoma; however, a negative subdural tap does not rule it out. If the clinical condition of the patient is deteriorating, as manifested by a very tense fontanelle, rapid pulse, respiration, shock, retinal hemorrhage, and pupillary changes, a cerebral arteriogram should be done. If the subdural tap alleviates the patient's condition, further careful observation is then permissible. If there is no amelioration of symptoms or if the tap is negative, then one should consider the possibility of the hematoma in a location other than the convexity. One of the most useful views in the arteriograms is the submentovertex one, which permits the visualization of the entire periphery of the brain from the frontal region to the occipital.

Often an arteriogram is negative, but as the swelling decreases, a week or 10 days later a subdural hematoma of the chronic variety is found, because the brain of the newborn infant, in particular, atrophies, leaving behind a space that is occupied by a heavy xanthochromic fluid. It is because of the way in which the child's brain behaves that the physician should not become complacent, even if the patient's condition improves.

Repeated subdural taps should be done before discharge as well as electroencephalography and brain scan. If the child's recovery is slow and the tests appear to be positive or inconclusive, a repeated arteriogram

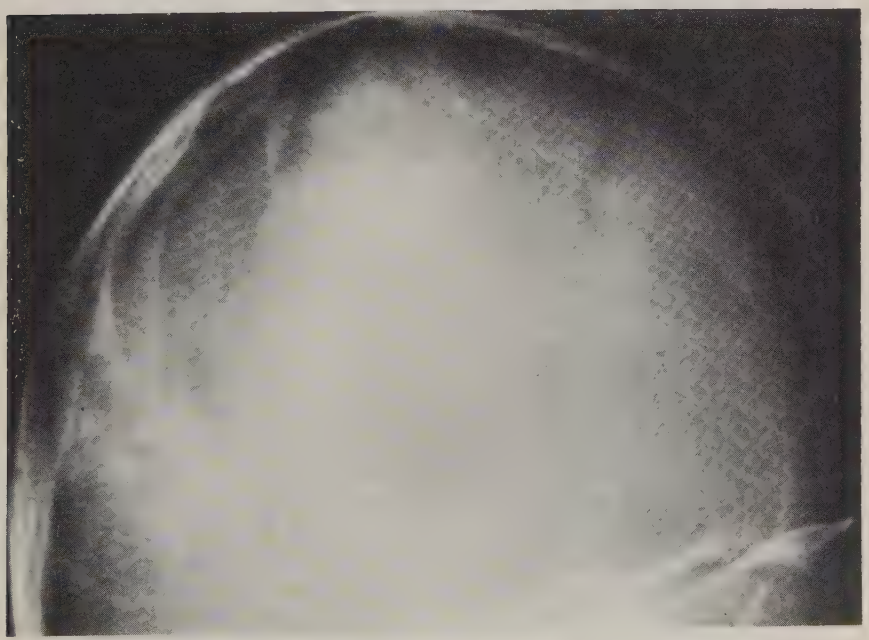

Figure 2. Growing skull fracture.

is indicated. It is catastrophic not to recognize the abnormality in time since the chronic subdural hematoma in children up to 9 years of age carries a very significant morbidity.

Head injury may be accompanied also by a subarachnoid hemorrhage, and a hydrocephalus may occur secondary to the bleeding in the subarachnoid space. Six patients in our series had such complications. If the hydrocephalus is of the communicating variety, demonstrated arteriographically, then a shunting procedure is the treatment of choice and it could be ventriculoatrial, ventriculo-peritoneal, or lumbo-peritoneal.

Chronic Subdural Hematoma

Of all the clinical entities which result from craniocerebral injury, none is more complicated in etiology or pathogenesis, and none presents a more challenging problem to either the clinician or the surgeon, than the subdural hematoma.

The neuroradiologic diagnosis presents no problems since the angiographic picture is very characteristic. Generally the child has sustained a head injury that may have been severe, with loss of consciousness, vomiting, or convulsions, but the injury does not have to have been of great magnitude. In fact, perhaps no sign of trauma will be found. The child may be placed in the hospital because of irritability, but without localizing signs, and discharged a few days later, clinically sound and eating well. In the follow-up the physician may notice that the normal neuromuscular development of the child has slowed down in the sense that, perhaps, he is not able to hold his head as before or has stopped rolling around on the bed. The cephalic circumference may be increasing in size. On the other hand, the child could be smiling and even have a sunken fontanelle, but on routine check-up the cephalic circumference may be found to be increasing at a fast rate. This should alert the pediatrician to the existence of a chronic subdural hematoma. As a rule, in head injury, a large head with a sunken fontanel indicates a chronic subdural hematoma until proven otherwise.

Periodic evaluation of the eyegrounds will show progressive optic disk pallor. Bilateral sixth nerve palsy or paresis may be present. Subdural taps will reveal large amounts of xanthochromic fluid. Electroencephalography may help in showing the classic low voltage over the hematoma. Brain scan is recommended.

The chronic subdural hematoma has the tendency to be encapsulated, to be located over the convexity, interhemispheric or subtemporally, and most of the time beyond the reach of the needle's tip when subdural taps are done; therefore, when a subdural hematoma is suspected clinically, and then corroborated by a suspicious brain scan or electroencephalogram, cerebral arteriography is indicated. As a rule, once the hematoma is chronic, the spinal tap may or may not reveal xanthochromia. The plain x-ray of the skull may or may not show evidence of increasing intracranial pressure. If the hematoma is discovered early enough, repeated subdural taps may be the treatment of choice.

Many neurosurgeons believe in and even publish excellent results concerning the surgical treatment of the chronic subdural hematoma,

whether the treatment consists of burr holes and evacuation of the fluid, or craniotomy with stripping of the subdural membrane, basing their impression on a happy child with good appetite and sunken fontanelle. But unfortunately, for the physician this impression is an erroneous one. First of all, those who report a possible cure never repeat cerebral angiography or pneumoencephalogram after operation, and it has been demonstrated that 8 hours after successful surgery these children have a reaccumulation of subdural fluid.

The underlying pathology is not only the hematoma but the cerebral atrophy that follows the cerebral contusion and/or the chronic cerebral compression. On the basis of the printed material we are of the firm conviction that a craniotomy with stripping of the subdural membrane is nothing else but a surgical exercise. The placement of a subduroperitoneal shunt, *with no valve*, is the treatment of choice and it is less traumatic. The arteriographic appearances of the hematomas are shown in Figure 4. In the acute phases, the accumulation of blood in the subdural space can be seen arteriographically as space-filling defect over the entire convexity of the brain. Figure 4 illustrates the convexity-filling defect seen in the anteroposterior view of the arteriogram and the defect along the interhemispheral surface and under the temporal lobe. It should also be noted that the collection of blood compresses the brain and thereby causes cortical and perforating vessels to cluster together. In children, the distribution of the hematoma is better seen over the entire

Figure 3. Arteriographic appearance of arteries caught in a fracture (leptomeningeal cyst).

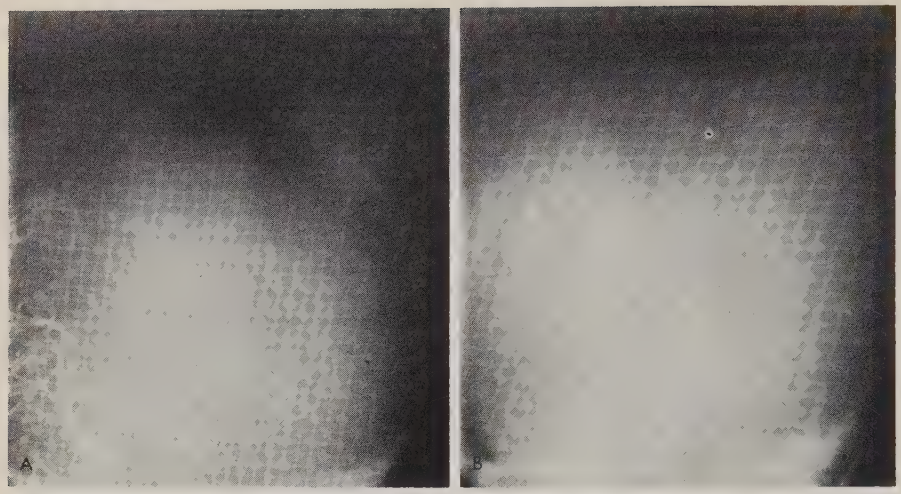

Figure 4. Chronic subdural hematoma. *A*, Arteriographic appearance. The convexity filling-defect is seen in the anteroposterior view (*B*). The defect is also seen along the interhemispheral surface and under the temporal lobe.

hemisphere in the lateral arterial and venous phase. In Figure 5 we see that in the lateral view the arteries fail to reach the inner table of the skull. Figure 6 shows brain atrophy secondary to a chronic subdural hematoma. In this chronic subdural hematoma, the lateral arteriogram is absolutely characteristic in that it reveals a picture of a "hanging brain" suspended by the veins. Normally, the veins in the posterior portion of the brain runs against the main stream of the sagittal sinus. In the chronic subdural hematoma they are completely vertical in their course.

Epidural Hematoma

Lineal or depressed skull fractures may cause epidural hematomas. When the fracture occurs at the vertex (see Fig. 7), it may produce laceration of the superior sagittal sinus, and this anatomical structure may be compressed to the point that the blood flow through the lumen is markedly decreased or completely obstructed. It should be remarked that the diploe alone without a laceration of the sagittal sinus may provide a large amount of bleeding to produce a similar effect as in the actual laceration of the superior sagittal sinus.

In infants and newborns, epidural hematomas usually result from accumulation of blood originating from the diploe. Very seldom, if ever, is the middle meningeal artery responsible for the epidural hematoma. The epidural hematomas in children are self limited due to the fact that the dura represents the inner periosteum of the bone of the skull, and it is fused with the outer periosteum at the suture lines. In many instances the origin of the bleeding over the superior sagittal sinus may be located in the middle third, yet the hematoma may be more extensive over the anterior third, displacing the sinus downward.

The anterior third is the most dissectable portion of the sagittal sinus. Bleeding may occur into the subgaleal space as well, through the lineal

fracture; the epidural hematomas decompress themselves in this fashion, alleviating or avoiding cerebral compression or brain shift.

Plain x-rays should alert the pediatrician when the fracture line is over the superior, sagittal, or lateral sinuses. If there is a depressed fracture over either one of these anatomical structures, cerebral angiography should be done prior to any surgical procedures, since a large rent may be present (Fig. 7).

When the hematoma occurs in the posterior fossa the arteriogram may show direct as well as indirect evidences of hematoma formation. Indirect evidence is seen in the form of acute hydrocephalus, with bowing of the anterior cerebral artery, and elevation and stretching of the middle cerebral artery. The distance between the anterior and middle cerebral arteries is increased, with lateral displacement of the lenticulostriated arteries. Upward displacement of the cerebellum gives direct evidence of hematoma formation. The superior cerebellar arteries run, in the lateral films, above the territory of the posterior cerebral arteries. The basilar artery is displaced anteriorly against the clivus. The course of the superior cerebellar arteries indicates upward herniation of the cerebellum. This upward herniation also can produce alteration in the course to the vein of Rosenthal and the vein of Galen, producing verticalization of these blood vessels from their regular horizontal courses.

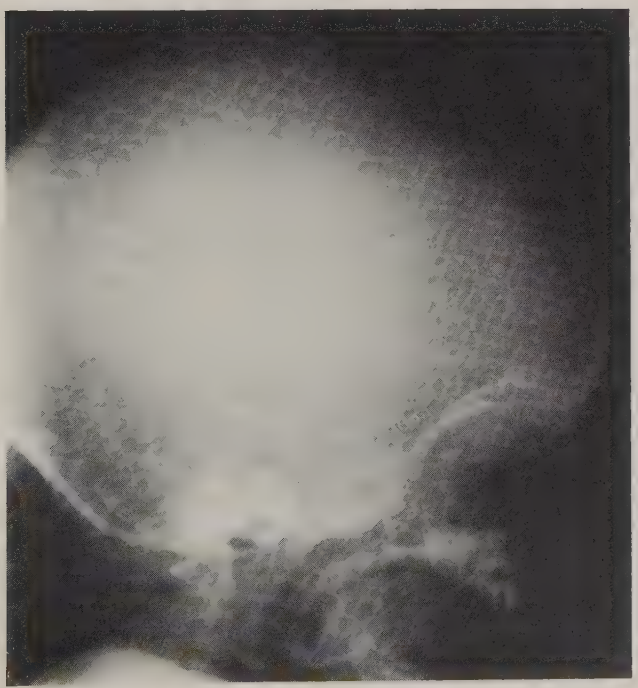

Figure 5. Chronic subdural hematoma. In the lateral view, the arteries fail to reach the inner table of the skull.

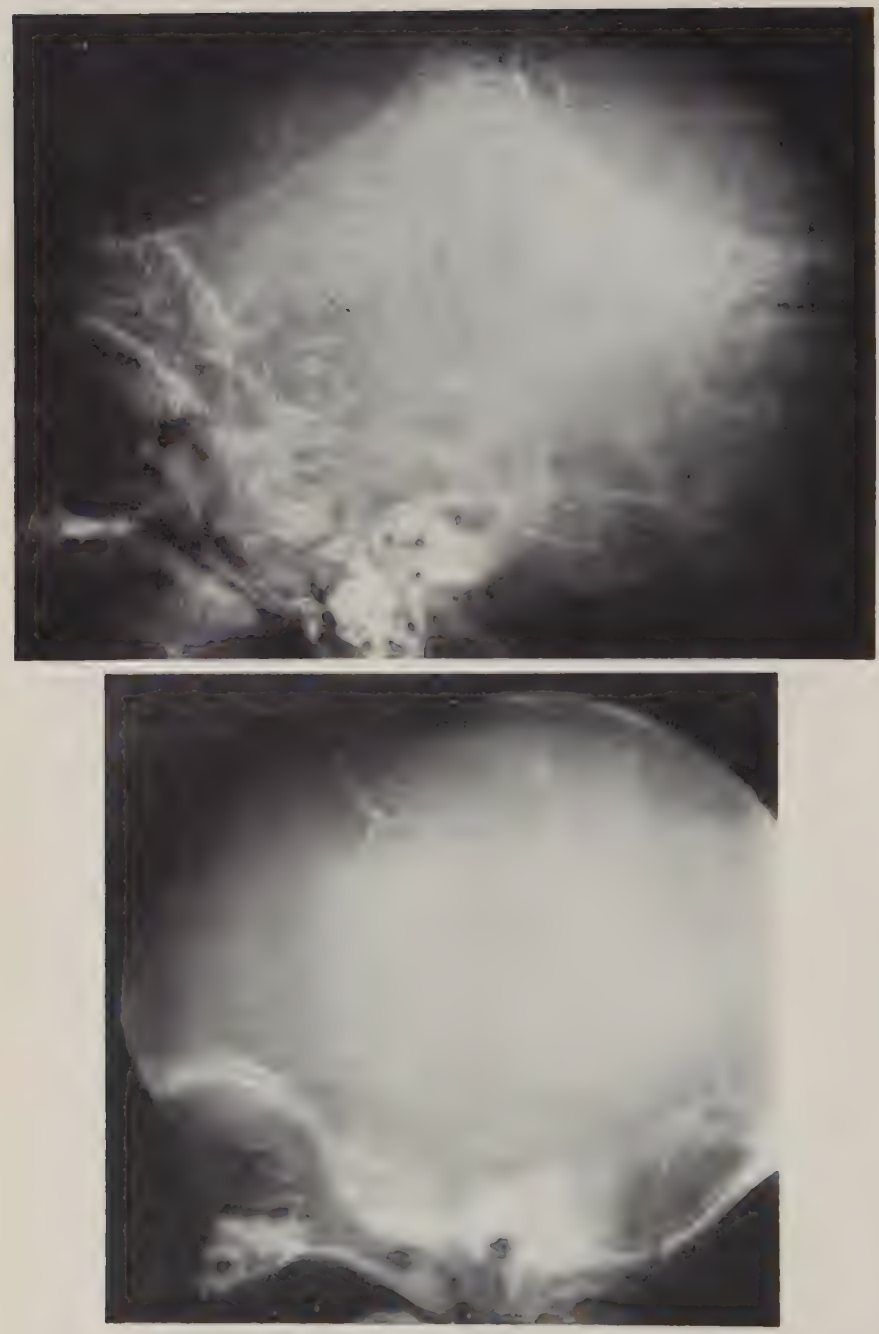

Figure 6. Brain atrophy secondary to chronic subdural hematoma. The brain is "suspended" by the veins.

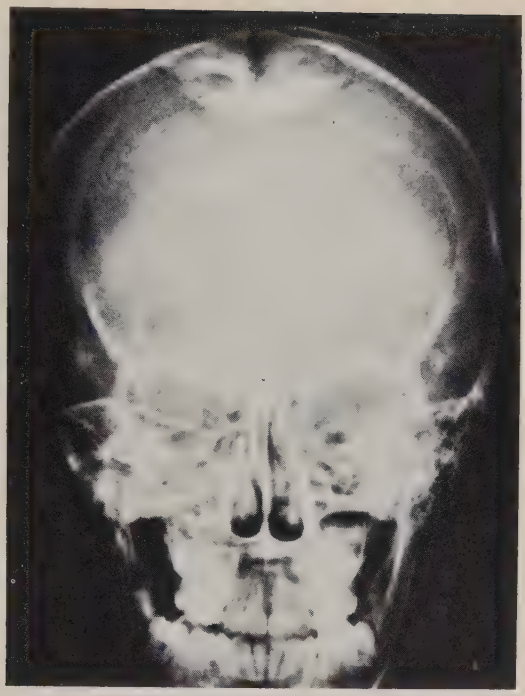

Figure 7. "En bloc" depressed fracture.

CEREBRAL CONCUSSION AND CONTUSION

The term cerebral concussion has been used with wide and indefinite connotations, as it will mean that there is little to no damage to the brain. Most authors agree that there is transient loss of consciousness secondary to head trauma. This period of unconsciousness usually is of less than 5 minutes' duration. The child may become irritable, sleepy, and have changes in alimentary habits. Recovery from a cerebral concussion is almost always complete.

Gurdjian et al. have made some observations on the mechanism of the brain concussion. They reported that acceleration and deceleration produces an increase in the intracranial pressure which in turn produces a detrimental effect in the brain stem. The cerebral concussion may or may not be accompanied by a lineal skull fracture.

Cerebral contusion and laceration result from craniocerebral injury of the greatest magnitude. The surface of the brain becomes bluish and the cerebral cortex becomes hemorrhagic and edematous. These cerebral changes occur most often as a result of direct trauma over a given region in the head. Cortical damage occurs more commonly at the poles of the frontal and temporal lobes. Contusions of these two poles may represent a counter-coup phenomenon rather than the result of direct trauma over the frontal or temporal region. Contusion of the frontal pole results in posterior displacement of the whole complex of the anterior cerebral ar-

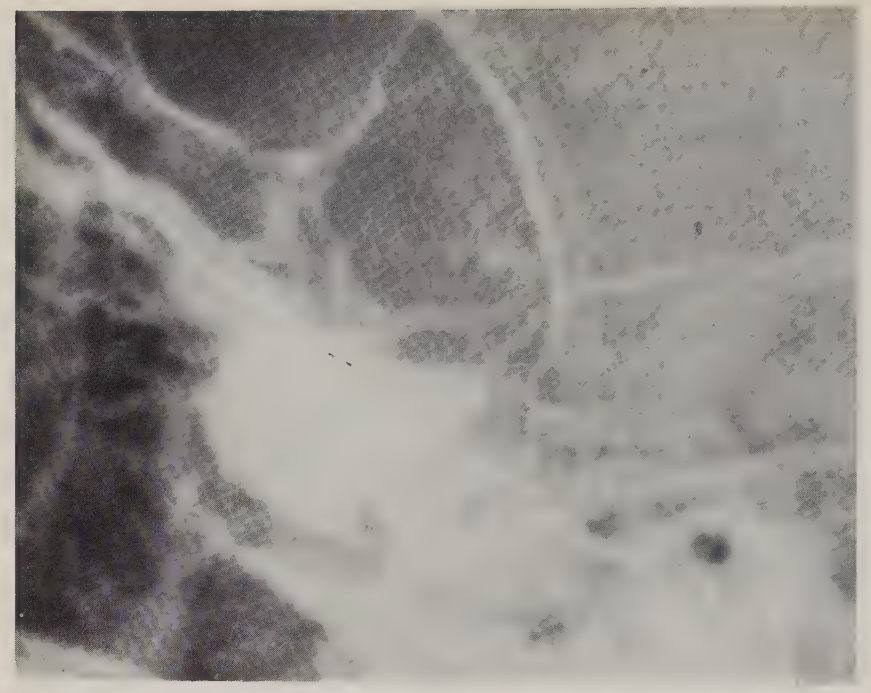

Figure 8. Cavernous sinus fistula.

tery and may cause rapid herniation with or without cerebral incarceration under the falx cerebri. If there is a massive cerebral edema, transtentorial herniation of the temporal lobes can be diagnosed by the notching which is formed in the posterior communicating artery. Compression of the third cranial nerve is responsible for the clinical picture of dilated pupil on the herniated side and paralysis of the extraocular movements. Once the herniation occurs, the posterior cerebral arteries may be kinked at the edge of the tentorium, and thus the blood supply of the occipital lobe will be in jeopardy and infarction in the inferior aspect of the temporal lobe and occipital lobe may result. This will, of course, produce more cerebral edema, which will aggravate the clinical picture.

A temporal lobe contusion may be evident by elevation of the middle cerebral artery, which may or may not be accompanied with shifting of the anterior cerebral artery, internal cerebral vein, or both.

Parietal lobe contusion is caused by direct injury to the parietal region, often as a result of the use of forceps during delivery. A swelling of the parietal lobe rarely will produce transtentorial herniation. Angiographically, the contusion is best seen in the lateral views. One can see that the ascendent frontoparietal arteries are spread apart by the cerebral edema.

Contusion of the parietal lobes should be treated conservatively with hypertonic agents. The most commonly contused lobe in the infant is the temporal lobe. If the diagnosis of lobar contusion is made in a child who

shows clinical evidence of deterioration, the neurosurgeon may decide to resect the contused lobe. This surgical procedure carries a negligible mortality. On many occasions the skull fractures are accompanied by cranial bruits and pulsatile exophthalmus that can be associated with complete ophthalmoplegia. When this occurs arteriovenous fistula of the cavernous sinus should be ruled out (Fig. 8).

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Trauma to the Neck Region

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Perhaps no other anatomic area contains a greater number and variety of important, closely packed structures than the neck. Consequently, any neck trauma may potentially injure an essential component of the vascular, neurologic, respiratory, digestive, or skeletal systems.

Undoubtedly serious injuries to the neck would be even more common were it not for the protection afforded posteriorly by the cervical spine and thick paracervical muscles and anteriorly by the shoulders and mandible, which shield the flexed neck from anticipated injury.[8]

The magnitude of underlying injury is often obscured by an innocent appearing neck wound. Similarly, a neck injury may be overlooked initially because concomitant head or chest trauma diverts the examiner's attention. Failure to appreciate the seriousness of the neck injury may than lead to tragic consequences, including delayed hemorrhage, airway obstruction, deep neck infections, and cerebral infarction.

TYPES OF INJURY

Exclusive of cervical spine and muscular injuries, the most frequent type of neck trauma requiring surgical consultation is the penetrating variety, owing to gunshot, shotgun, or stab wounds.

Blunt neck trauma usually results from an automobile accident and it often produces the most difficult and challenging injuries to manage. The resultant skeletal and neurological damage is usually immediately recognized but too often other serious soft tissue injuries are overlooked.

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Iatrogenic neck injuries are the least common but their incidence is probably on the rise, primarily because of various misadventures with subclavian catheters and the increased use of cuffed endotracheal and tracheostomy tubes with resultant tracheal stenoses. Recurrent nerve injuries during thyroid operations and esophageal perforations during endoscopy hopefully are on the decline.

INITIAL MANAGEMENT

All patients with head and neck trauma, especially those who are unconscious, should be treated as though their neck was broken until this possibility can be definitely ruled out by the cervical x-ray films.[19, 20] The head should be continuously supported in a neutral position to avoid an inadvertent severing of the spinal cord.

The first priority in the management of patients with neck trauma is the establishment of an adequate airway. Initially this may require only the clearing of blood, secretions, and dentures; traction on the tongue; and/or insertion of an oral airway. At other times an endotracheal tube must be inserted immediately.

While the primary importance of a patient's airway cannot be overemphasized, the existence of other injuries must be considered. For example, endotracheal intubation should be undertaken with great caution if there is a possibility of an associated injury to the cervical spine. In this situation, hyperextension of the neck in an attempt to visualize the larynx may result in irreparable damage to the cervical spinal cord. When this conflict of priorities exists, nasotracheal intubation or a temporary cricothyroid tracheostomy may be a safer alternative.

If the patient has sustained severe trauma to the larynx or trachea, an endotracheal tube often cannot be passed and an airway must then be established by means of tracheostomy below the area of injury or an endotracheal tube inserted through the entry hole of the penetrating laryngeal or tracheal wound.[3]

Late airway obstruction may result from the development of an expanding hematoma beneath the pretracheal fascia, and this possibility should always be kept in mind.

Once an adequate airway has been established, proper ventilation must be assured. If necessary, ventilation is supported by whatever means available, e.g., mouth-to-mouth respiration, Ambu bag, or mechanical respirator. The chest is examined to be sure that no hemothorax or pneumothorax exists, which would require the immediate insertion of a thoracostomy tube.

In most instances, bleeding from penetrating neck wounds can be controlled with direct pressure. The use of circumferential dressings or blind clamping into a neck wound are condemned.[22]

CASE NO. 1

A 28 year old man sustained a "clothesline" injury to the neck when he drove his snowmobile under a chain fence. He walked into the emergency room of the

Figure 1. Patient who sustained a "clothesline" injury to the neck while driving his snowmobile under a chain fence.

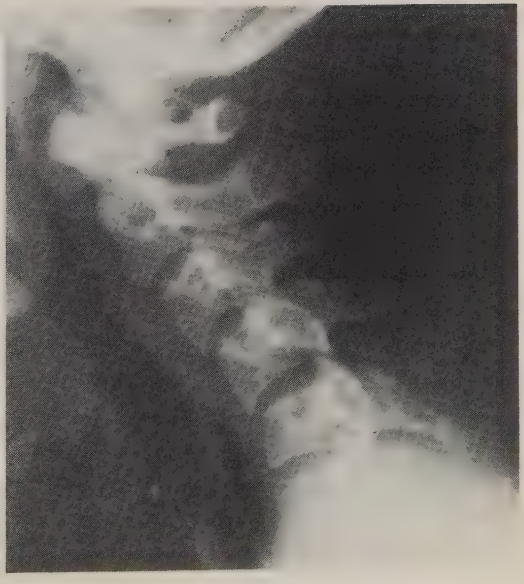

Figure 2. Lateral x-rays of the neck, revealing a subluxation of the body of C_4 over C_5. Note the fractured hyoid bone and the retrotracheal air collection.

Loyola University Hospital several minutes after the accident, complaining of pain on swallowing and swelling about the neck (Fig. 1).

Fortunately, the patient had no initial respiratory distress so emergency neck x-rays were obtained prior to performing a tracheostomy (Fig. 2). The x-rays revealed a subluxation of C_4–C_5.

The cervical subluxation was treated immediately with traction by means of Crutchfield tongs inserted in the emergency department under local anesthesia. Ten days later anterior cervical fusion and a collar provided permanent stability.

Surgical exploration the night of injury revealed a comminuted fracture of the hyoid bone and a tear in the thyrohyoid membrane with posterior displacement of the larynx. These injuries were repaired and a laryngeal stent was inserted.

Comment. If this patient had experienced immediate respiratory distress, and the possibility of a cervical spine injury had not been entertained, hyperextension of the neck in an attempt to insert an endotracheal tube or perform a tracheostomy, could well have produced a permanent spinal cord injury.

DETECTION OF INJURIES

Once the initial management of the patient's injury has been completed, a thorough history and physical examination are performed. Special attention is directed toward detection of the following types of injuries.

Vascular Injuries

Vascular injuries may be easily recognized in the presence of shock, active bleeding, or large, expanding hematomas. Equally frequent however, is the serious vascular injury which is masked by the absence of these findings as well as by an innocent-appearing neck wound.

Examination should include palpation of the carotid, superficial temporal, and upper extremity pulses and comparison of the quality of the pulse and blood pressure on both sides of the body. Diminished or absent pulses are good evidence of a vascular injury; but often the pulses are normal in the presence of a significant injury, because the artery is only partially severed and a good flow of blood continues into the distal vessel. In addition, if the injured artery has no readily palpable point for evaluating distal pulses (e.g., internal carotid, vertebral, or thyrocervical trunk) or if the injury is to the venous system, evaluation of pulses is of little benefit.

Auscultation is an extremely important yet frequently overlooked mode of diagnosis. A systolic bruit is a characteristic finding in patients with a partial artery injury and a resultant false aneurysm, whereas a continuous bruit suggests the presence of a traumatic arteriovenous fistula.

CASE NO. 2

An 18 year old boy sustained a gunshot wound of the left neck. On physical examination, the bullet was palpable in the right axilla. The patient was

hospitalized and observed in the intensive care unit of a local Chicago hospital for 2 days and then discharged.

He was seen the following day in the emergency room of Cook County Hospital complaining of a "roaring" in his ears which kept him awake all night. On examination, a loud continuous bruit was heard over the sternum. The patient was admitted to the hospital and a selective innominate arteriogram was performed (Fig. 3) and revealed an innominate arteriovenous fistula.

The fistula was repaired through a median sternotomy and the patient had an uneventful postoperative course.

Comment. In this case, an arterial injury was initially overlooked, presumably because previous examiners neglected to listen for the presence of a bruit.

Vertebral artery injuries are especially elusive but should be suspected whenever: (1) neurologic deficit develops in the centers supplied by the basilar vertebral system (brain stem, cerebellum, eighth nerve); (2) carotid artery compression fails to slow the bleeding or obliterate the bruit coming from a wound whose trajectory crosses the posterolateral aspect of the neck; (3) massive bleeding accompanies a cervical transverse process fracture.[11]

The fact that the vertebral artery travels through the transverse processes of the cervical vertebrae accounts for the failure of compression to control bleeding from this vessel, and greatly increases the difficulty of surgical exposure.

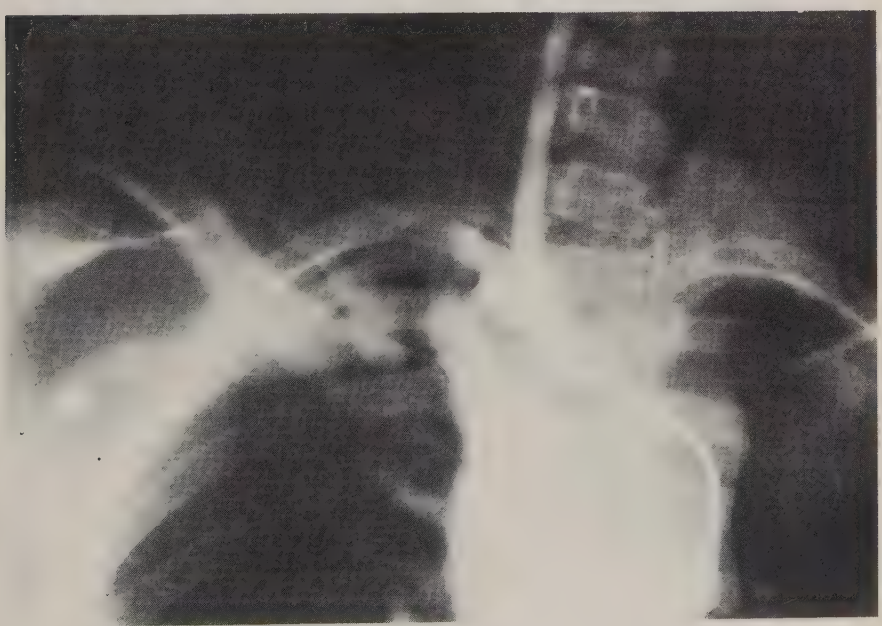

Figure 3. Selective innominate arteriogram revealing an innominate arteriovenous fistula.

CASE NO. 3

A 31 year old woman entered Cook County Hospital in profound shock. Bright red blood was rushing from a 1 cm. stab wound in the posterior triangle of the left side of the neck.

Blood pressure, on admission, was unobtainable, and the pulse was weak and rapid. The bleeding from the neck wound was inadequately controlled with pressure, and the patient received rapid transfusion of 8 units of whole blood and 3 units of clinical dextran. During resuscitation, she had two cardiac arrests which were managed with closed cardiac massage.

The patient was rushed to the operating room where a neck exploration revealed a 90 per cent transection of the vertebral artery between the transverse processes of the third and fourth cervical vertebrae. The artery was approached by removing these two transverse processes, permitting ligation proximally and distally.

The patient had another cardiac arrest in the operating room and died 6 hours postoperatively, never having regained consciousness.

Comment. This case emphasizes the lethal potential of the partially severed vertebral artery. Had this vessel been completely severed, hemorrhage might well have been minimal since the vessel would have been able to retract and constrict.

Carotid artery injuries are often suspected in penetrating wounds of the neck but they may also result from blunt trauma and should be considered in any patient with: (1) a hematoma in the upper anterior triangle of the neck, (2) Horner's syndrome, (3) transient ischemic attacks, (4) loss of consciousness following a lucid interval, or (5) the development of hemiplegia.[15, 27] These findings may often be absent initially and then appear days to weeks later as a slowly developing intramural hematoma obliterates the lumen of the carotid artery.

Neurologic Injuries

The most serious of the neurologic injuries is quadriplegia resulting from severance of the cervical spinal cord. Also trauma to the root of the neck may produce brachial plexus damage, usually in association with vascular injury.

The vagus, recurrent laryngeal, spinal accessory, hypoglossal, phrenic, and cervical sympathetic nerves are all subject to injury secondary to neck trauma. Vagus and recurrent laryngeal nerve injuries may produce hoarseness and are confirmed by observation of an immobile vocal cord on direct or indirect laryngoscopy.

The spinal accessory and hypoglossal nerves are checked by assessing the motor function of the trapezius and tongue muscles respectively. A cervical sympathetic nerve injury is recognized by the presence of an ipsilateral Horner's syndrome.

Perhaps the most important part of the complete neurologic examination is the determination of the level of consciousness. If an accurate and descriptive recording of the patient's initial level of consciousness is made, subtle changes can be detected which may suggest an intracranial injury or an expanding intramural hematoma of the extracranial carotid artery.

Figure 4. Patient who sustained a fractured larynx in an automobile accident. Note the visible neck swelling, which represents subcutaneous emphysema.

Trachea and Larynx

Injuries to the trachea and larynx are suspected in patients with dyspnea, hoarseness, stridor, or subcutaneous emphysema (Fig. 4).

Fractures of the larynx or trachea can often be palpated through the intact overlying skin.

Pharynx and Esophagus

Severe pain and dysphagia are characteristic symptoms of a perforation of the pharynx or esophagus. Physical examination should include digital palpation of the oropharynx and hypopharynx as well as careful palpation of the neck in an attempt to feel subcutaneous crepitation.

Pulmonary and Cardiac

An intrathoracic injury should always be suspected in patients with penetrating wounds of the neck.[10] Often the path of a knife blade is difficult to determine, and we have seen stab wounds of the neck produce not only pneumothorax and hemothorax, but even cardiac tamponade.

Initial physical examination and chest x-ray films may be normal but once the patient is taken to the operating room and anesthetized, the picture can change dramatically. Positive pressure ventilation may produce a tension pneumothorax through a previously unrecognized small stab wound of the lung parenchyma. Unless the problem is immediately appreciated and treated, the patient may expire in a matter of minutes (Fig. 5).

Figure 5. Tension pneumothorax seen in posteroanterior chest film of a patient who sustained a stab wound of the right neck. Note marked shift of the mediastinum to the left.

DIAGNOSTIC PROCEDURES

X-RAYS. Anteroposterior and lateral films of the neck are essential in order to look for displacement of the trachea, narrowing of the airway, subcutaneous emphysema, cervical fractures and gunshot fragments.

Posteroanterior and lateral chest film may reveal widening of the superior mediastinum, suggesting a great vessel injury, hemothorax, or pneumothorax.

ESOPHOGRAM. A barium esophogram can help to delineate an injury to the cervical or thoracic esophagus, if extravasation is noted. However, a normal study does not exclude an esophageal injury.[24]

ENDOSCOPY. Direct laryngoscopy, bronchoscopy, and esophagoscopy are extremely helpful in locating injuries to the larynx, trachea and esophagus.[26] When conditions permit, these procedures should be performed in the operating room immediately prior to neck exploration.

Occasionally, an endoscopic procedure will initiate active bleeding from neck wounds. Therefore, the patient is prepared and draped for neck exploration prior to starting these procedures, so that if uncontrollable bleeding ensues, endoscopy can be terminated and the neck immediately explored.[8]

ARTERIOGRAPHY. When considering arteriography we arbitrarily categorize injuries into three zones: Zone I, below the sternal notch; Zone II, between the sternal notch, and the angle of the mandible; and Zone III, above the angle of the mandible (Fig. 6).[18]

Injuries extending below the sternal notch (Zone I) should be fully evaluated with a preoperative retrograde femoral aortic archogram. This

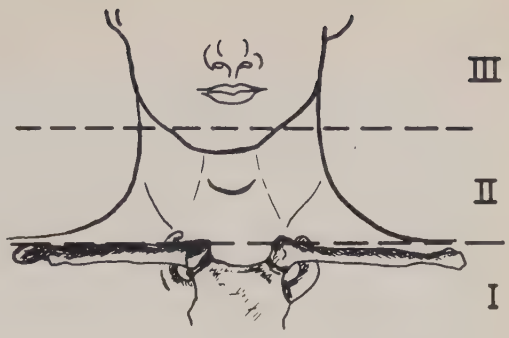

Figure 6. Zones of injury in neck trauma.

allows good visualization of the innominate, common carotid, subclavian, and vertebral arteries and is invaluable in helping to determine the appropriate operative approach.

Injuries between the sternal notch and the angle of the mandible (Zone II) are easily explored and arteriography is usually not performed for these injuries.

In patients with injuries extending above the angle of the mandible (Zone III) a percutaneous carotid arteriogram is performed. This gives specific information about the status of the internal carotid artery and the intracerebral circulation. Arterial repair is difficult in these injuries and the arteriogram helps to determine the feasibility of repair and the likely consequences of ligation (Fig. 7).

Figure 7. Carotid arteriogram revealing a false aneurysm of the carotid bifurcation in a patient who was stabbed in the neck above the angle of the mandible.

SURGICAL EXPLORATION

Decision

Although articles advocating a selective approach to neck injuries continue to be published,[22-24] our experience and the overwhelming experience of others convinces us that all patients with penetrating wounds of the neck should undergo surgical exploration.[1, 11, 14, 16, 26, 30] We have repeatedly found significant injuries in patients who had otherwise innocent appearing neck wounds.

Long lacerations or small wounds which appear to involve only the skin are first inspected to determine the depth of penetration. If it can be clearly established that the platysma muscle is intact, transfer to the operating room for repair may not be necessary. Once platysma muscle penetration is identified, the patient is prepared for transfer to the operating room. Probing of neck wounds is not justified.

Preparation

In the operating room a large bore nasogastric tube or Ewald tube is inserted. This helps to prevent gastric dilatation and aspiration as well as aiding in the identification of the esophagus during surgical exploration.

All explorations are performed under general anesthesia[12, 13] and a large sterile field is prepared and draped to include the entire anterior chest and neck as high as the mastoid eminence. This allows the surgeon to readily extend his incision into the chest for proximal control of vascular injuries when this is necessary.

Exposure

The neck is usually explored through a long incision paralleling the anterior border of the sternocleidomastoid muscle. This approach provides excellent exposure and access to all the important structures of the mid neck. In addition, it can be readily extended upward to gain access to the internal carotid artery or jugular vein. Inferiorly it may be extended down the midline of the sternum for easy access to the great vessels of the thorax. The fascia over the anterior border of the sternocleidomastoid muscle is incised from the sternal notch to near the mastoid prominence. The muscle is then retracted laterally to expose the strap muscles and the carotid and jugular vessels. The omohyoid muscle is usually severed. The sternothyroid and sternohyoid muscles can be retracted medially or, if necessary, divided transversely to allow access to the larynx, thyroid, trachea, or esophagus. If both sides of the neck require exploration, a transverse collar incision is occasionally used.

Results

Routine exploration of penetrating neck wounds has decreased mortality rates by eliminating such late complications as delayed hemorrhage, airway obstructions, and deep neck infections.[11] Furthermore, even negative explorations, which should have no mortality, result in a lower incidence of wound infection by close adherence to the principles of debridement, hemostasis, and drainage.[16]

MANAGEMENT OF SPECIFIC NECK INJURIES

Vascular

Injuries to the innominate, vertebral, subclavian, common carotid, and internal carotid arteries may be repaired by either lateral arteriorrhaphy, resection, and end to end anastomosis, vein patch, or vein by-pass. Smaller arteries of the neck can be ligated. Injuries to the internal carotid artery near the base of the skull pose a very difficult problem in repair, and active bleeding will usually require ligation of the proximal artery and packing of the distal stump.

If a vein must be used for repair, the saphenous vein is preferred because of its thick wall. The use of expendable veins from the neck, e.g., the external jugular vein, often results in ballooning of the vein when it is subjected to arterial pressure. Occasionally, if the injury involves the great vessels coming from the aortic arch, a Dacron graft is used, but in general, synthetic grafts should be avoided in the contaminated wounds usually encountered in trauma.

In patients with an established neurologic deficit associated with a carotid injury, repair of the vessel is probably inadvisable because of the likelihood of converting an ischemic cerebral infarction to a hemorrhagic one with progressive neurologic deterioration.[6, 18] Ligation in this situation is preferable.

Our experience suggests that when no neurologic deficit exists, the use of internal shunts during carotid artery repair is unnecessary.[6, 18] We have encountered no difficulties from clamping the carotid artery for periods long enough to achieve adequate repair. The use of temporary internal and external shunts may add reassurance to those familiar with their use.[4, 19]

Any injured vein may be ligated but small tangential tears are usually repaired. Pressure should be immediately applied to all venous injuries to avoid air embolism.[22, 24]

Injuries to the intrathoracic innominate, proximal right subclavian, and common carotid arteries as well as the innominate veins are best exposed through a median sternotomy.

The midportion of the subclavian arteries and the origins of the vertebral arteries are exposed through a supraclavicular incision with resection of the medial half of the clavicle.

The intrathoracic portion of the left subclavian artery is approached through the left third intercostal space anteriorly.[5]

Neurologic

Injuries to any of the important nerves of the neck are easily overlooked unless they have been highly suspected and searched for prior to operation.

Important cranial or brachial plexus nerves are carefully debrided and repaired with fine arterial silk sutures approximating the perineurium. A large nerve deficit may be bridged with the use of an expendable sensory nerve, e.g., the greater auricular nerve.

Injuries to the spinal cord above the third cervical segment cause

complete respiratory paralysis and are rapidly fatal unless immediate resuscitation is instituted. Lower injuries to the cervical spinal cord produce lesser degrees of respiratory paralysis along with variable motor and sensory deficits, up to and including complete quadriplegia.

Most of these injuries are associated with cervical spine fractures or fracture-dislocations. These fractures are usually stabilized by means of Crutchfield tongs and cervical traction. Surgical exploration is reserved for patients with fracture dislocations which cannot be reduced with traction, and also for patients with progressive neurologic findings or the presence of bony fragments or missiles within the spinal canal.

Trachea and Larynx

The immediate management of penetrating wounds of the trachea and larynx may require the insertion of a tracheostomy tube either through the area of injury or preferably below it.[21] This life-saving procedure is often so dramatic that it diverts attention away from the area of injury. However, unless laryngeal and tracheal injuries are primarily repaired, late strictures and stenoses will result.[7, 19]

Minor hematomas, lacerations, and undisplaced fractures of the larynx and trachea caused by blunt trauma require close observation, broad spectrum antibiotics, and high humidity.

More serious lacerations or contusions which result in the loss of mucosa must be treated with split-thickness skin grafts held in place by an internal stent for 2 to 3 weeks.[13, 28] The stents may be constructed simply by inserting foam rubber within a finger cot.[9] The stent and the graft are then held in place by No. 24 stainless steel wires which pass through the stent and the larynx and are brought out each side of the neck.

Severely displaced fractures of the larynx require open reduction, mucosal repair or grafting, and internal stenting often for 6 to 9 months.[17]

Large avulsion injuries of the trachea may require replacement with Marlex mesh.[3]

Pharynx and Esophagus

Injuries to the pharynx and esophagus are repaired by two-layer closure (full thickness chromic catgut plus silk) after adequate debridement. The wound is drained for 3 to 4 days and the patient is placed on appropriate broad-spectrum antibiotics. A Levine tube is left in place and used for feeding purposes when necessary.

Large defects in the cervical esophagus may be difficult to approximate. Rarely both ends of esophagus may be brought out the wound as cutaneous esophagostomies. Staged reconstruction of the cervical esophagus is then accomplished over several months.[29]

A small injury to the esophagus can sometimes be identified only by filling the wound with saline and giving positive pressure breathing per mask. Bubbles will then be seen to rise from the point of injury.

An overlooked esophageal injury may lead to neck abscesses and mediastinitis, with lethal consequences. Adequate drainage may prevent their development even when repair has not been accomplished.

Miscellaneous

Injuries to the thyroid and salivary glands are treated by debridement, thorough hemostasis, and neck drainage.

Injuries to the thoracic duct may occur in penetrating wounds of the base of the neck. If surgical exploration reveals a milky fluid accumulating in the surgical field, a careful search must be made for the duct which is then ligated above and below the area of injury with nonabsorbable suture. An unrecognized injury to the thoracic duct will result in a chylous fistula or chylothorax.[2]

CONCLUSIONS

Trauma to the neck frequently results in multiple, regional organ injury, and in addition poses serious threats to vital structures in the head and thorax. Initial measures should be directed toward preserving the integrity of the cervical spinal cord and establishing and maintaining an adequate airway. Appropriate treatment is facilitated by a thorough history and physical examination conducted systematically to detect specific injuries. When feasible, surgery should be preceded by careful radiologic and endoscopic studies to identify the location of missiles and to establish the integrity of the major arteries, air and food passages. Surgical exploration of all penetrating neck wounds is a sound principle which merits continued adherence.

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Experience in the Operative Management of Penetrating Injuries of the Neck

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The appropriate treatment of penetrating injuries of the neck remains a topic of controversy in the surgical literature. Suggestions regarding appropriate management range from the conservative to the urgent.[1-4] Experience in the management of soft tissue injuries of the neck at the University of Chicago has substantiated our conviction that all penetrating neck wounds should be explored at the time of clinical presentation. Conservative or nonoperative management is reserved for minor superficial injuries only. A review of our current consecutive experience with penetrating neck wounds is presented, outlining the basis of our operative treatment.

PATIENT MATERIAL

Between 1964 and 1971, 70 patients with penetrating injuries of the neck were admitted to the University of Chicago Hospitals and Clinics. The youngest patient was 5 years old, the oldest was 61 years of age. The mean age was 30 years. There were 63 male and 7 female patients. Thirty-eight patients (54 per cent) sustained gunshot wounds to the neck, while 28 patients (40 per cent) had knife-inflicted injuries. Four patients' injuries were the result of shattered-glass lacerations sustained in automobile accidents. In 13 per cent of the patients, two or more associated injuries within the neck were present, while in 51 per cent of the patients the neck injury was one of multiple bodily injuries. The distribution of injuries to 54 anatomic structures of the neck is outlined in Table 1.

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Table 1. *Anatomic Distribution of Neck Injuries Within the Viceral Compartment and the Carotid Sheath*

Arteries		8
Common carotid	4	
Subclavian	2	
Internal carotid	2	
Veins		14
Internal jugular	9	
External jugular	3	
Other	2	
Thyroid		5
Trachea		6
Cricoid cartilage		1
Esophagus		9
Spinal cord		9
Brachial plexus		2
TOTAL		54

PATIENT MANAGEMENT

At the present time, all patients admitted with a penetrating injury to the neck, i.e., *any injury which has penetrated deep to the platysma muscle*, undergo neck exploration at the time of presentation. All patients are admitted to the primary care of a general surgeon. When multiple injuries are present, these are jointly managed under the guidance of the various specialty consultants. Decisions as to priority of management are thus coordinated through one team of surgeons.

Resuscitative management in the accident room must be especially aggressive in the case of neck injuries. Actual injury to the trachea or larynx may seriously complicate the initial airway management. Anything less than aggressive appropriate therapy may result in serious and possibly irreversible morbidity. Often, intubation or tracheostomy are life-saving if the airway cannot be maintained by spontaneous or mask ventilation. Profound hemorrhage with significant hypotension may complicate penetrating neck injuries. Rapid correction with whole blood (preferably type-specific) is often essential if management is to be successful.

Blind clamping of bleeding vessels is strongly condemned. A clean pressure dressing will usually suffice to control brisk hemorrhage. Tamponade also serves to obviate sucking venous injuries and prevents air embolism.

Should the patient's condition remain quite stable, a chest x-ray should be obtained prior to operation. Also, if a bullet wound does not reveal a point of exit, i.e., the trajectory remains obscure, x-rays of the neck may act as a guide to appropriate exploration.

If hemothorax or pneumothorax are suspected, but the patient is too

unstable to undergo diagnostic radiography, needle aspiration of either hemithorax may be accomplished with impunity. Occasionally, "blind" tube thoracostomy on the basis of high suspicion alone has been life-saving.

Operative Treatment

The objectives of neck exploration are two-fold: visualization of the structures of the carotid sheath and exploration of the contents of the "visceral compartment," i.e., trachea, thyroid, esophagus, etc. In addition, hemostasis, irrigation, debridement, and reconstruction of associated muscular and fascial injuries is accomplished. All lesions are explored via a transverse cervical incision at the level of the mid neck or, in the case of lacerations, at the level of the injury. Auxiliary or supplementary incisions are employed as needed.

The following modalities were employed in the management of specific injuries in our series:

One injury to the common carotid artery was ligated below the superior thyroid artery. Despite anticipated collateral circulation, ligation resulted in hemiparesis. Two lacerations of the carotid artery were repaired by simple suture. By-pass shunts were employed in the management of irreparable carotid injuries, i.e., either to achieve end-to-end re-anastomosis or, in one case, to re-establish continuity by means of an autogenous vein graft.

One injury to the subclavian artery was managed by ligation while another was managed by primary repair. The latter required a simultaneous approach via anterior thoracotomy in addition to the low transverse neck incision. Although an initially employed Dacron prosthesis underwent late thrombosis, this was subsequently replaced without consequence.

Superficial venous injuries were treated by simple ligation. Similar management was employed in the case of deep venous injuries where, in five instances, the internal jugular vein was unilaterally ligated. Three patients with internal jugular vein damage underwent primary suture closure while one patient underwent a patch graft repair.

Injuries to the thyroid were treated by debridement and hemostasis. In one gunshot injury, lobectomy was dictated by the extent of the damage. Although we presently employ tracheostomy in all cases, tracheal injuries were handled by three methods. In two cases, primary repair of the laceration was achieved without the use of tracheostomy. However, four other injuries did require the use of a tracheostomy. In one patient a tracheal stent was employed without primary repair. Although this required replacement after being coughed out, the resulting healing was perfectly satisfactory.

One patient who sustained a laceration of the cricoid cartilage required re-exploration when a hematoma developed in the neck shortly following endotracheal extubation and associated laryngospasm. At re-exploration, the hematoma was evacuated, hemostasis was achieved, and tracheostomy was performed. Recovery was uneventful thereafter.

Nine injuries of the esophagus were managed by two-layer suture

repair supplemented by the placement of a soft rubber drain. Despite normal recovery and complete absence of clinical signs, two patients manifested evidence of extravasation of radiopaque contrast (Gastrografin) on esophagogram. Injuries to the spinal cord were treated by decompression laminectomy in three patients (33 per cent). Three patients did not undergo any operative treatment for their central nervous system injury. Three additional patients were transferred to other hospitals prior to exploration.

Results

There were two deaths in the postoperative period. Both patients sustained irreparable injuries to the central nervous system.

For those patients requiring soft tissue repair only, there was an average hospital stay of 8 days. There were no deaths in this group. Two patients had complications (10 per cent) and only two patients required transfusion. In the group in which serious visceral or vascular injuries were encountered, two deaths resulted from central nervous system injury. The complication rate was 46 per cent. The hospital stay averaged 10 days and nearly 50 per cent of the patients required blood transfusion. Overall, 24.5 per cent of the patients received blood. Eighty-nine per cent of the patients were treated with antibiotics.

Two patients who did not undergo exploration were subsequently found to have brachial plexus injuries secondary to penetrating trauma. In this series, no damage to the recurrent laryngeal nerves was encountered as a direct result of penetrating injury.

There were no complications of carotid arterial repair when a bypass conduit was employed or when simple suture repair was required. In contrast, ligation of major vessels resulted in serious sequelae. One patient whose carotid artery was ligated suffered a resulting hemiplegia, while the patient whose subclavian artery was ligated was left with a weakened ischemic left upper extremity. When a Dacron prosthesis was employed to replace an injured segment of the subclavian artery, thrombosis occurred, requiring subsequent re-exploration. Replacement with a similar graft was successful.

The results of venous repair was generally satisfactory. One patient who sustained simultaneous internal jugular vein injury and phenothiazine overdose subsequently developed contralateral hemiplegia following internal jugular venous ligation. Otherwise there was one case in which a hematoma required re-operation. All thyroid repairs or resections were uncomplicated.

Except in the case in which tracheal stent required replacement, all injuries to the trachea healed primarily. Some residual hoarseness was evident in the patient who was treated with the stent. One case of recurrent laryngeal nerve palsy occurred following repair of an iatrogenic injury to the membranous trachea incurred at the time of emergency tracheostomy (see Case No. 2).

Two of the 9 patients who underwent esophageal repair demonstrated radiographic extravasation of contrast material on esophagogram (Fig. 3). Neither patient developed clinical evidence of a leak, i.e., drainage, fever, abscess.

All 3 patients who underwent decompression laminectomy manifested improvement of their paresis within the immediate postoperative period. One patient developed a superficial wound infection. Of the 3 patients who did not undergo laminectomy, 1 died of increased intracranial pressure with a midline cerebral shift demonstrated by carotid angiography. Another patient with combined central nervous and carotid artery injuries died after development of a consumption coagulopathy. The possibility that this patient received mismatched blood could not be proved.

Two additional patients were admitted to the hospital with lacerations of the neck. Both were thought to have superficial injuries and neither underwent exploration. Subsequent brachial plexus palsies were evident in both patients with permanent loss of function in the affected extremity.

Twenty of the 70 patients treated for penetrating injuries to the neck, had damage limited to muscle, fascia, and superficial unnamed vessels. Following debridement, hemostasis, and primary repair, 1 patient developed a hematoma, while a second patient had a wound infection. Ninety per cent of the patients made an uneventful recovery following primary cosmetic repair.

In contrast, of the 24 patients who did not undergo operation and primary reconstruction, 3 patients developed wound hematomas. Two other patients required drainage of wound infection. The nonoperative morbidity was 22 per cent.

The following case studies illustrate several of the described features in the management of visceral and vascular neck injuries.

CASE REPORTS

CASE 1

A 34 year old negro man sustained a gunshot wound to the neck, inferior to and to the right of the thyroid cartilage. His neck was quite swollen and tender but there was no neurologic or pulse deficit. Cervical x-rays demonstrated a fragmented bullet at the base of the neck above the pulmonary apex. The patient was taken to the operating room and underwent a low transverse incision. Visual examination revealed destruction of the right thyroid lobe, and a through-and-through penetration of the right common carotid artery. The arterial lesion was clamped and bypassed with a No. 16 Bardic catheter, the traumatized area was resected, and an anterior jugular vein autologous graft replacement was sewn into place. Following removal of the shunt, the distal arteriotomy was re-opened to evacuate a thrombus. The patient did well postoperatively. A patent graft is seen on arch aortogram nine days later (Figure 1).

CASE 2

A 43 year old negro man sustained a slash injury to his neck. He was seen in the emergency room in a state of profound shock, unresponsive to pain. Tracheostomy was accomplished in the emergency room. Transfusions were delivered through three separate conduits. While being resuscitated, the patient was brought to the operating room and underwent neck exploration. Severe strap and sternomastoid muscle injury, carotid sheath hematoma, multiple large arterial and venous hemorrhages, and an iatrogenic posterior tracheal wall perfo-

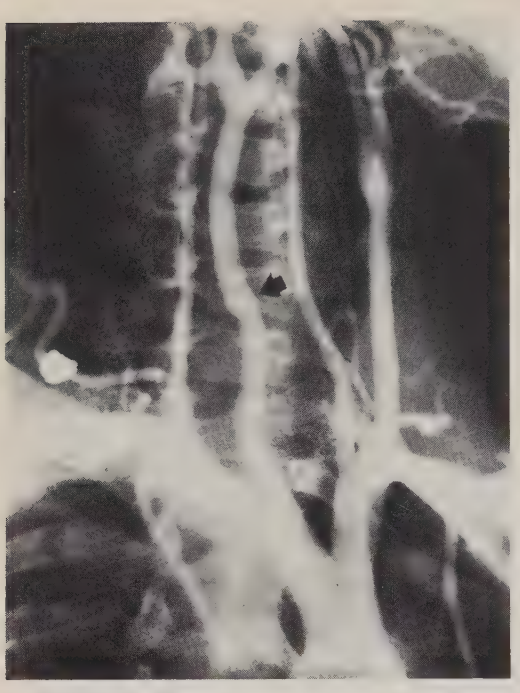

Figure 1. Aortic arch anteriogram demonstrates normal architecture 10 days following vein-graft prosthetic repair of the right common carotid artery.

Figure 2. Laryngogram 9 days following repair of the membranous trachea. There was clinical and laryngoscopic evidence of left vocal cord paresis. The true vocal cord is edematous with asymmetric distortion of the left pyriform sinus. Recovery was complete.

ration secondary to the tracheostomy were found . The carotid sheath was explored and no abnormalities were encountered. Bleeding vessels were ligated, the left recurrent laryngeal nerve was intact and traced, and the esophagus was noted to be bruised. The trachea was dissected from the esophagus and closed in two layers. A latex rubber drain was left at the site. The patient did well postoperatively without central nervous system difficulties. Laryngogram, however, demonstrated left vocal cord paralysis. The patient was discharged 10 days after operation without a tracheostomy and in good condition. By 4 months following operation, partial function of the left vocal cord had been regained (Fig. 2).

Case 3

A 61 year old negro man entered the hospital with a gunshot wound to the lateral left neck. On admission he had a swollen tender neck. There was no neurologic or pulse deficit. A chest x-ray film was unremarkable. Blood was present in the hypopharynx. At exploration a lacerated left internal jugular vein was ligated and a left lateral esophageal lesion closed in two layers and drained. Postoperatively the patient did well. Radiographic evidence of an esophageal leak was treated successfully with clear liquid diet. No clinical leakage ensued. The patient was discharged in good condition 7 days following operation (Fig. 3).

DISCUSSION

The surgical literature reflects continuing controversy over the primary management of penetrating neck injuries. Ideally, a statistically

Figure 3. Esophagogram performed 1 week after repair of esophageal laceration. Note the presence of contrast material outside the normal lumen.

valid comparative study of management should be available to answer this problem. In the absence of such a study, the following several factors related to the question of exploration might be considered in formulating a rationale of treatment:

1. Generally speaking, the morbidity of operative treatment when serious injuries are present remains low.[2]

2. The morbidity of operative treatment for minor injuries is less than half the morbidity of nonoperative treatment, as seen in the present series.[1]

3. The treatment of complications resulting from uncorrected injury is more complicated and less successful than the primary repair of serious trauma.[4]

Although the number of neck injuries in the community served by this University has remained constant over the past 5 years, the ratio of missile to knife injuries continues to rise yearly. As gunshot wounds of the neck generally result in greater damage than do knife injuries, it may be expected that penetrating neck injury will continue to result in more serious damage than in years past.

The morbidity and mortality associated with soft tissue injuries of the neck reflect the magnitude of the injury as well as the expertise of primary management. Naturally, superficial injuries of the neck require little attention. The essence of the debate concerning proper primary management reflects the feeling that the operative cost to patients with musculofascial injury alone outweighs the benefit of immediate operation in the patients in whom serious visceral or vascular damage is encountered.

We are of the opinion that the prevention of hematoma formation and infection, combined with primary cosmetic repair, are all in the patient's best interest even when serious injury is not encountered at primary neck exploration. Certainly, delayed management of serious injuries can only lead to serious complications. As an example, in our own experience two patients who did not undergo exploration were left with permanent peripheral nerve damage. Weaver[1] has similarly reported serious complications resulting from a policy of non-operation.

Of the 46 patients who underwent operative treatment, the majority required repair of major structures within the visceral compartment or within the carotid sheath. The remaining patients required hemostasis and musculofascial repair, benefiting as well from functional and cosmetic reconstruction (Table 2).

Table 2. *Results of Treatment of Penetrating Injuries of the Neck*

	SERIES	OPERATIVE	NONOPERATIVE
Number	70	46	24
Per cent	100	56	44
Hospital stay (days)	8	10	4
Morbidity	27%	30%	29%
Mortality	2.9%	4.3%	0
Transfusion	24%	28%	12.5%

It should be noted that diagnostic chest x-ray examination helped in the detection of trauma-related abnormalities in 13 of these 46 patients (28 per cent), confirming the usefulness of this procedure when the patient's condition is stable.

We are convinced that in the management of penetrating injuries to the soft tissues of the neck, the policy of immediate exploration and repair seems the logical course. The results of operation reflect better overall treatment for these patients than the results of any form of observation.

Expectant triage should be limited to patients with obvious superficial lacerations and to the occasional patient in whom more serious injuries demand the priority of immediate attention.

Most of the serious consequences of conservative management are the result of failure to appreciate the true depth of a penetrating injury at the time of initial inspection. It is our belief that in any laceration in which the superficial nature of the injury is not immediately obvious, the assessment of platysma penetration should be performed under local anesthesia *in the operating room,* where evaluation can be made definitively. Errors in defining the actual depth of injury (with resulting serious consequences) may thus be obviated, with subsequent treatment of the patient by appropriate neck exploration or simple debridement.

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Burns of the Head and Neck

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During the 6½ years between July 1965 and December 1971, 2601 patients were treated for acute thermal injuries at the Sumner L. Koch Burn Unit, Cook County Hospital, in Chicago. Eight hundred and eighty-one of these patients sustained burns about the face and neck.

The patients with burns about the face and neck ranged in age from 1 to 92 years. There were 608 males (69 per cent) and 273 females (31 per cent), compared to 1700 (65 per cent) males and 901 (35 per cent) females in the entire group. The extent of injury ranged from 1 to 100 per cent; one patient was admitted with a complete body burn after being rescued from a burning rubbish pile where he had been thrown in an attempted murder. There was a 31 per cent mortality (276 of 881) compared to an 18 per cent mortality for the entire group of patients admitted during this same period. Burning clothing was the most common cause of injury, accounting for 320 accidents. Scalds, explosions, industrial accidents, and smoking in bed were the other major causes.

Morbidity associated with burns about the face and neck relates to the important organs and structures of the area, the eyes, ears, nose, mouth, and the specialized skin of the face and neck. The effect of a thermal injury on each of these structures will be considered in this article, as well as the problem of the pulmonary thermal injury that occurs frequently in patients with burns about the face and neck.

THERMAL PULMONARY INJURY

A thermal pulmonary injury can be extremely difficult to diagnose and treat. Injury to the pulmonary tree should be suspected in a patient with burns about the face and neck, especially when the injury occurred in a closed area or where there is a likelihood that hot air, steam, smoke, or toxic gases may have been inhaled. In a patient with burns about the face and neck, if there are burns of the palate and oropharynx, one can be reasonably sure that some degree of pulmonary injury has occurred.

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Findings will vary according to the extent and time after injury. In minor pulmonary injuries there are rarely symptoms or findings for several hours to days. The most common findings will be an increased respiratory rate, occurring between the second and sixth days, followed by radiographic findings of parenchymal consolidation. If the blood gases remain within normal ranges, oxygen by mask or catheter will be all that is required, unless sepsis appears to be significant, requiring the use of antibiotics.

In patients with more extensive pulmonary thermal injuries, symptoms and findings usually occur earlier, are more severe, and often require varied and prolonged therapy. Early findings may include an increase in respiratory rate, gasping, distant breath sounds, wheezing, and rales. While radiographic findings are rarely abnormal early after injury, abnormal blood gas findings do occur and suggest a serious injury. With any combination of the above symptoms, in a patient whose history and physical findings suggest a pulmonary injury, early treatment with 100 per cent oxygen and high humidity is indicated. Frequent tracheal aspiration is usually required. The aspirate or coughed sputum should be carefully examined for soot or other foreign material. Most patients will respond to oxygen and humidity; however, on occasion certain symptoms and findings will persist and require other therapy. Early and repeated bronchoscopy may be the only means to adequately manage thick sooty secretions.

Severe wheezing and distant breath sounds are suggestive of severe tracheobronchial edema and/or bronchospasm. The latter is probably not common since treatment with high humidity and oxygen usually relieves these symptoms. When symptoms and findings persist after therapy with oxygen and high humidity, it is unusual to find a patient that will respond to the usual bronchodilators. However, large doses of steroids (1.5 to 3 gm. of hydrocortisone) will give permanent or temporary relief for several hours in most patients.

Persistent decreased tissue oxygenation along with a low arterial Po_2 and elevated arterial CO_2 calls for efforts to increase the transport of oxygen across the alveolar membrane. This can best be accomplished by the use of positive pressure, delivered by a ventilator through an endotracheal tube.

Only a few years ago when our knowledge of blood gas evaluations was less sophisticated and our information regarding respirators was less advanced, tracheostomy was performed much more often than it is today in patients with thermal injuries. Tracheostomy should be performed in the burned patient on the same indications as any other surgical patient, i.e., for upper airway obstruction, inability to handle secretions, and when prolonged tracheal intubation would be required to maintain adequate tissue oxygenation.

Pneumonia, atelectasis, abscess formation, and pulmonary emboli also occur in patients with thermal injuries about the face and neck. These conditions are frequently a result of the thermal injury per se, rather than the fact the face and neck were involved in the injury.

BURNS OF THE EYES

A burn to the eye viscera proper is an unusual occurrence. In reviewing over 1000 consecutive patients with thermal injuries, none was found to have a conjunctional or globe injury. (We have treated 3 patients who sustained thermal injuries to the eyes from hot chemicals.) However, burns of the eyelids commonly occur in patients with thermal injuries about the face and neck. In the 881 patients reviewed for the purpose of this article, 81 per cent had some degree of eyelid involvement.

The management of a patient with burns of the eyelids should be based upon the extent and depth of the local injury, associated injuries, and the general plan of therapy. It is illogical to treat all eyelid burns in the same manner without strongly considering the extent and depth of the individual injury.

Our current management is based upon the knowledge that most burns of the eyelids are partial-thickness in depth and will heal spontaneously without significant scarring. With deep partial-thickness burns, in some patients scarring and contractures will occur. In others, this does not occur. When contracture and lid eversion is noted early after injury, the use of a properly applied pressure dressing will usually prevent the development of a severe contracture. On occasion, a contracture will be so significant that tarsorraphy or suturing the lids closed will be required until healing is complete (3 to 4 months). When this is necessary, meticulous cleansing and the use of ointments will prevent the formation of irritating crusts and exudates.

In the case of full-thickness injuries, the likelihood and problems of scarring are much greater. Contracture with lid eversion is likely to occur earlier, with exposure of the cornea, and the hazards of corneal ulceration. Efforts to keep the lids closed by the use of tarsorraphy or suturing must be considered early after injury. In addition, techniques to obtain early eschar removal and permanent closure with autografts should be utilized. Surgical excision of a full-thickness eschar of the eyelid is usually not practiced. Frequent (4 or 5 times daily) changes of dressings are usually utilized to aid in eschar removal. This technique will allow the superficial, the intermediate, and some of the deep dermal burns to heal spontaneously while the full-thickness eschar is being removed. By an aggressive approach, using frequent dressing changes, full-thickness burns of the eyelids can often be ready for grafting by the tenth to twelfth days. In patients with extensive injuries, their general condition will usually allow the taking of small grafts at this time. On occasion, full-thickness wounds of the eyelids will be ready for autografting before the patient's condition permits or before donor sites are available. The use of homografts or heterografts as biological dressings is extremely helpful during this time. They are applied over the raw or granulating surfaces and changed every 2 to 4 days. In addition to serving as a temporary dressing, the homograft and heterograft will prevent the loss of fluids from the wound, aid in debridement of eschar remnants, and reduce bacterial growth.

Following the application of autografts to full-thickness burn wounds of the eyelids, some type of dressing is usually applied. The type and duration of the dressing depends upon several factors; the most important of these is whether or not the patient has a tarsorraphy, or the eyelids are sutured closed. If so, the dressing does not have to be designed to restrict motion, and in certain patients with the eyelids immobilized, no dressings are used. Immobilization of the eyelids after grafting should continue until the grafts are well taken and there is no further chance of significant contracture.

With proper meticulous care during the acute phase of patients with eyelid burns, the need for reconstruction will be minimized. When contracture and eyelid eversion does occur after a thermal injury early correction is occasionally indicated to prevent corneal ulceration. If corneal ulceration is not a problem when there is contracture of the eyelid, it is preferable to wait until the scar has matured and the contracture is no longer progressing.

BURNS OF THE NOSE

Fortunately, full-thickness burns of the nose rarely occur. This is probably due in part to the excellent vascularity of the area. However, partial thickness burns of the nose occur quite commonly (about 70 per cent) in patients with burns about the face and neck. Meticuluous care of a partial-thickness burn of the nose will not only expedite healing, it will also prevent the conversion of an epidermal burn to a dermal depth and a deep partial-thickness wound to a full-thickness injury. Treatment of the burned nose should be individualized, as the response to injury tends to vary considerably from patient to patient. Early after injury, the burned nose should be thoroughly cleansed and the loose tissue removed. Depending upon the depth and exudative response, burns of the nose may be managed by frequent cleansing with soap and water, the use of topical antibacterials, or coverage with biological dressings.

The use of topical antibacterial agents (Sulfamylon or silver nitrate solution) should be reserved for the deep partial-thickness wounds, or in the case of full-thickness burns where there are extensive areas elsewhere, and early eschar removal is not desirable.

Biological dressings (homografts or heterografts) are used in deep partial-thickness injuries to minimize pain, reduce bacterial growth, and to prevent the conversion of these particular injuries to full thickness. Either homografts or heterografts may be applied to deep partial wounds immediately or at any stage prior to re-epithelialization.

One of the more important times to apply a biological dressing to the deep partial-thickness burn wound is during and immediately after the separation of crust or eschar. At this time the epithelial elements that participate in the re-epithelialization of the wound are most liable to further injury from trauma and sepsis. The use of either homograft or heterograft at this time will protect the remnants of sweat glands and hair follicles and allow the re-epithelialization to occur.

The management of full-thickness burns of the nose, like full-

thickness burns in other important areas of the body, deserves individual consideration and care. Each situation will be different and the management should be based on the patient's age, extent of injury, associated injuries about the head and neck, and the etiology of the injury. As a general policy we avoid the use of topical antibacterial drugs on full-thickness burns of the nose unless there are extensive associated burn injuries where burn wound sepsis is a potential problem. We have also avoided the practice of early surgical excision of full-thickness burns of the nose. Our concern in regards to early surgical excision has been to damage of the underlying cartilage, with a resultant deformity that is a significant cosmetic problem.

To minimize the conversion of a full-thickness burn of the nose to a greater depth than the presenting injury and/or damage to cartilage we have frequently used the technique of almost continuous saline soaks to the burned nose. This method of treatment keeps the crust and eschar soft, and allows for early eschar separation often as early as 10 to 12 days after injury. With this slow separation of the eschar, granulation tissue has usually developed, with the appearance of a relatively healthy wound. Depending upon the detailed appearance of the wound, a biological dressing may be utilized for a few days, or an autograft may be applied on the day of complete eschar separation. It has been our practice to apply a biological dressing to any granulating wound of the nose if there is any suggestion of purulent exudate or remnants of residual dermis. The biological dressing will decrease the bacterial growth and usually reduce the exudate. They will also aid in removing remnants of dermis that may interfere with the take of autografts. In addition, the take of a biological dressing is quite indicative of what can be expected from the take of an autograft. In essence the biological dressing can help prepare and test a granulating wound for receptivity of autografting.

Although the skin of the nose is thick and well vascularized, protecting the underlying cartilage, occasional deep partial-thickness burns of the nose will damage the cartilage and result in some loss. This will usually result in a significant deformity requiring reconstructive surgery.

BURNS OF THE EARS

Unlike the skin of the nose, the skin of the ear is thin and delicate. Therefore, the treatment of the burned ear will differ. One of the most common complications in patients with burns of the ears is the involvement of the cartilage. This may occur in both partial and full thickness injuries. This problem has been referred to as chondritis and perichondritis. These terms are indicative of an infectious process and the actual pathological process may be only a thermal necrosis of the cartilage. Because of prolific bacterial growth in the area of the burned tissue, secondary bacterial growth commonly occurs. The bacterial problem is probably not primary in most cases.

Chondritis or perichondritis of the auricular cartilage is usually diagnosed by complaints of pain in the ears. Even in the early stages the ears are usually swollen, inflamed (warm and discolored), and tender. In some

patients, with proper therapy there will be a fairly prompt resolution of this process with few or no residual problems. (Proper therapy is the systemic use of the maximal recommended dose of synthetic penicillin, since the majority of the wounds culture coagulase-positive Staph. aureus as the only or primary organism.) The majority of patients that develop the early signs of thermal necrosis (chondritis or perichondritis) of the auricular cartilage will progress to a stage of fluctuation; this is indicative of liquefaction of the cartilage or a purulent septic process. When fluctuation develops there is fairly general agreement that adequate drainage (in addition to systemic antibiotics) is basic therapy. Some feel that not only is adequate drainage essential, but suggest the removal of the cartilage. This recommendation is based upon the fact that drainage usually persists until the cartilage is removed or is completely liquefied. In either situation the result is a shrivelled ear, with a poor cosmetic appearance, that is difficult to reconstruct.

The best treatment for thermal necrosis of the auricular cartilage is prevention. Recent advancements in treating the burn wound have been effective in reducing the incidence of this problem. With the use of topical antibacterial agents and biological dressings the incidence has been reduced from 34 per cent in over 1200 patients in whom topical antibacterial agents or biological dressings were not used, to 12 per cent in patients in whom either topical antibacterials and/or biological dressings were used.

Since many burns of the ears involve only the upper margin, small areas of full or deep partial-thickness wounds occur. If they are full-thickness in depth, management with biological dressings without autografting will usually result in a satisfactory permanent closure. A significant advantage of this technique is the management of the patient without hospitalization since the biological dressing may be changed as frequently as needed on an outpatient basis. However, when autografts are required elsewhere, permanent closure of small full-thickness burn wounds of the ears may be expedited by applying autografts.

Reconstruction of the burned ear following loss of cartilage or skin is a difficult and special problem. If hair growth has not been seriously disturbed, many severely deformed ears may be adequately covered with hair from the adjacent areas.

BURNS OF THE SCALP

Like all areas of the head and neck, the scalp has an excellent blood supply. In addition the scalp is protected from thermal injury by a covering of hair (present in most patients). Nevertheless, full-thickness injury occurs more frequently than one would expect. The frequency of full-thickness burns of the scalp is probably due to the large numbers of hair follicles and sweat glands in the area, which makes cleanliness difficult to maintain.

It is important to initiate early appropriate therapy to a burned scalp in order to obtain optimal comfort and healing. Basic care consists of a thorough cleansing with soap and water and shaving of all hair-bearing

areas. This might meet with resistance in some patients and families; however, failure to do so may result in conversion of partial-thickness wounds to full-thickness and delay healing of wounds of all depths.

After cleansing and shaving, topical antibacterial agents and biological dressings may be indicated. In general, we have used topical antibacterial agents in treating deep partial-thickness injuries to prevent their conversion to full-thickness. Biological dressings have been used in all stages of partial-thickness injuries, although they have been used most commonly after eschar separation to protect the epithelial element that might regenerate.

In full-thickness burn wounds of the scalp, topical antibacterial agents are indicated when there is an extensive injury and a potential for burn wound sepsis. Biological dressings have been used on the full-thickness burn wound after eschar separation. As previously mentioned, the use of biological dressings at this time reduces bacterial growth, aids in debridement of the wound, and tests for receptivity of autografts.

In isolated full-thickness burns of the scalp, we have occasionally performed early surgical excision—especially in electrical injuries and other full-thickness burns of the scalp that result in exposure of the skull. If the periosteum remains viable, local flaps may be utilized. If the cortical bone is exposed for even a few days, necrosis will usually occur. This means that at least the outer table must be removed, and a satisfactory bed obtained prior to autografting in some patients. In some situations, holes may be drilled in the exposed skull that will allow granulating tissue to grow through. When sufficient areas of granulation tissue have developed, autografts may be applied. As in treating any granulating wound, the use of biological dressings may be indicated.

In full-thickness burns of the scalp where there is significant loss of hair-bearing tissue, reconstructive surgery can play a major role in rehabilitation of the patient. The excision of scarred hairless areas and the rotation of hair-bearing flaps can result in marked cosmetic improvement in most patients. Many of our patients with loss of up to 50 per cent of the hair-bearing tissue have had rotation of flaps to achieve complete coverage with hair-bearing tissue.

BURNS OF THE NECK

Most burns of the neck occur anteriorly and laterally, and for this reason present a particular problem in management. The routine management of the burn wound of the neck area is not different or difficult, compared to other areas of the body. However, the prevention of contractures is difficult. Appropriate positioning started early after injury is a prime factor. The patient should be instructed in the methods of keeping the neck extended. This includes sleeping or resting on pillows beneath the shoulders and neck, support of the head in an extended position while sitting, and support of the head in a somewhat extended position if the patient is prone on a Stryker frame or Circo-electric bed after wound closure. The use of collar supports and pressure type dressings is help-

ful in most patients. These devices are usually needed for months after injury, and their temporary discontinuance will often result in a rapidly developing deformity.

BURNS OF THE FACE

The need for an optimal appearance of one's face is an accepted fact in today's society. Appropriate therapy can minimize scarring and deformities in facial burns. Techniques of management are almost identical to those of the burned nose. Early coverage with moderately thick skin will tend to minimize contracture and scarring. In some patients that tend to develop excessive scarring, the use of pressure postoperatively and/or after healing will minimize this problem. Reconstructive or secondary surgery is frequently necessary in patients who have sustained deep partial-thickness or full-thickness burns of the face. It is essential to keep in mind the requirements for donor skin to cover a facial wound. The skin should be of similar characteristics and texture to the skin of the face. While it is impossible to duplicate, certain areas are more suitable than others, and should be utilized if possible.

SUMMARY

Burns of the head and neck represent a complex injury. The complexity is due to the variety of problems that may result from a thermal injury to this region. The vital structures such as the eyes, ears, nose and facial skin vary considerably in their response to injury and need for therapy. The pulmonary thermal injury that may accompany a burn of this region is also a complex problem that requires considerable effort to minimize morbidity and mortality.

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Management of Tumors of the Parotid Gland

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Tumors of the parotid gland are among the less commonly encountered new growths in the body, comprising from 0.2 to 0.6 per cent of all tumors and approximately 2 per cent of tumors of the head and neck. So uncommon are they that a country the size of Norway can anticipate a rate of 180 new parotid tumors per year.[21] The parotid gland is rarely the site of metastases from other malignancies, but when they are found, the primary site is usually a melanoma or an intraoral squamous cell carcinoma.[9, 26] Occasionally the parotid is involved in neoplastic diseases of the lymphatic system, notably the lymphomas.[28]

The majority of tumors of the parotid gland are seen in adults. Only rarely are they found in children, and in such cases they are usually benign hemangiomas.[27] Fortunately the soft consistency of hemangiomas facilitates their clinical differentiation from the less common solid tumors. The latter group in children are malignant in 50 per cent of cases.[22] In a review of 40 tumors of the parotid gland in children, malignant mixed tumors and mucoepidermoid carcinomas were the most common malignant lesions.[1]

In adults, benign tumors of the parotid predominate over malignant tumors in the ratio of three or four to one.[2, 4, 6] In our series of 197 parotid tumors at the Northwestern University-McGaw Medical Center, 144 were benign and 53 were malignant, a ratio of three to one (Table 1). While tumors of the parotid gland are about 12 times as frequent as tumors of the lesser salivary glands, the latter have a higher incidence of malignancy. The prognosis varies according to location, the best being for malignancies arising in the palatal area, and the worst for malignancies of the submandibular gland. The prognosis for malignancies of the parotid gland falls between these two groups.[11]

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Table 1. *Clinical Aspects of 197 Parotid Gland Tumors**

TUMOR TYPE	TOTAL NO.	MALE	FE-MALE	SIDE R	SIDE L	LOBE Deep/Super.	PAIN	PER CENT TOTAL
Benign Tumors	144	57	87	65	79	5 139	5	73%
Mixed tumors	93	29	64	42	51	4 89	4	47.2%
Papillary cystadenoma lymphomatosum	35	21	14	15	20	0 35	1	17.8%
Lipoma	9	5	4	4	5	0 9	0	4.5%
Cyst	5	1	4	3	2	0 5	0	2.5%
Oncocytoma	2	1	1	1	1	1 1	0	1.0%
Malignant Tumors	53	30	23	23	30	11 42	7	26.9%
Adenocarcinoma	20	11	9	11	9	5 15	5	10.2%
Solid adenocarcinoma	6	4	2	4	2	1 5	1	
Acinic cell carcinoma	2	2	0	1	1	1 1	0	
Adenoid cystic	12	5	7	6	6	3 9	4	
Mucoepidermoid carcinoma (High and Low Grade)	18	11	7	4	14	3 15	1	9.1%
Malignant mixed tumor	8	7	1	5	3	2 6	0	4.1%
Undifferentiated carcinoma	3	1	2	1	2	0 3	0	1.5%
Malignant lymphoma	2	0	2	0	2	1 1	1	1.0%
Squamous cell carcinoma	1	0	1	1	0	0 1	0	0.5%
Metastases to parotid	1	0	1	1	0	0 1	0	0.5%
Totals	197	87	110	88	109	16 181	12	

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Tumors which arise in the parotid gland affect right and left sides with equal frequency. They have a slightly higher incidence in women,[12, 16] with the exception of Warthin's tumor[8] (Table 1). Tumors arise in the superficial lobe of the parotid 10 times more often than in the deep lobe,[18] but the histologic types are identical.

HISTOLOGIC TYPES

Our experience with 197 parotid tumors (Table 1) is similar to that of other authors. Benign tumors constituted 73 per cent of the total, and malignant tumors, 27 per cent. In the benign group, mixed tumor (pleomorphic adenoma) was the most common type (65 per cent), followed by Warthin's tumor (papillary cystadenoma lymphomatosum) (24 per cent).

The most common cell type in the malignant group was the mucoepidermoid carcinoma (34 per cent). Most of these histologically were of low grade (14 of 18 in our series). Adenocarcinomas, taken as a group, constituted 38 per cent of the malignant tumors, but these were further divided into acinic cell carcinoma, adenoid cystic carcinoma, and solid adenocarcinoma (Table 1). Although our series revealed more malignant tumors in men than in women, larger series have reported women to outnumber men.[2, 12]

DIAGNOSIS

The typical tumor of the parotid gland is first noticed as a mass by the patient or friend. One hundred eighty-six tumors in our series presented in this manner. Inspection and palpation by the physician confirm the presence of a mass in the region of the parotid gland. Intraoral examination is essential, since tumors of the deep lobe of the parotid may project into the pharynx. Berdal and Hall[5] reported an incidence of 4.4 per cent of parotid gland tumors with parapharyngeal projections. An effort should be made to ascertain the rate of growth and the presence or absence of symptoms. In our series, 12 patients had pain. In 5 (3.4 per cent) of these the tumors were benign, and in 7 (13.2 per cent) they were malignant. Adenocarcinoma, especially cylindroma, was more commonly painful than were other tumors.

Our only 2 patients with facial palsy had cylindromas (adenocystic carcinomas). The association of pain and facial weakness with cylindroma has been noted previously.[2, 3] This combination of symptoms may be due to the propensity of this tumor to invade nerve tissue and migrate along perineural spaces.

There was a high degree of correlation between rapid growth and malignancy in our patients. Although the duration of symptoms was vague in some, the patient with a large mass of short duration was usually found to have a malignant tumor.

Most benign tumors are movable on examination. A fixed parotid tumor is suspicious of malignancy. In spite of the rare occurrence of facial palsy in association with benign parotid tumors,[15, 20, 30] the triad of demonstrable metastasis, fixation to or invasion of adjacent structures, and facial palsy is pathognomonic of malignant parotid neoplasm. Unfortunately, in the absence of these signs, there are no clinical findings which positively differentiate between benign and malignant tumors. The diagnosis must be made histologically.

While some authors have reported that sialography assists in the differentiation of benign from malignant lesions[10, 31] we have not had this experience, and do not routinely employ this aid; nor do we use needle biopsy for reasons which have been outlined by others.[24]

It is apparent that a definite preoperative diagnosis is not possible in most patients with parotid gland tumors. It is the surgeon's responsibility, therefore, after confirmation of the presence of a tumor, to counsel the patient that this lesion may be malignant and that a thorough operation might require the sacrifice of the facial nerve.

SURGICAL TECHNIQUE

Simply stated, the treatment of tumors of the parotid gland is surgical excision. This has two objectives: (1) complete extirpation of the tumor and (2) preservation of the facial nerve whenever possible. Radiation therapy and chemotherapy usually have only palliative effects.

The technique that we favor is essentially that used by many other

surgeons. It requires wide exposure of the field with identification of the facial nerve. General endotracheal anesthesia is mandatory and in selected patients, the addition of hypotensive anesthesia has at times facilitated the dissection and expedited the surgery. Curariform agents are not employed, as they may interfere with identification of the facial nerve. Draping of the field must leave exposed the entire ipsilateral side of the face so that movement of the facial musculature may be observed. A nerve stimulator is used. The surgeon may free the seventh nerve by beginning peripherally and tracing the branches centripetally or may identify the main trunk at the stylomastoid foramen and proceed peripherally. In our view, there is no superiority of one technique over the other. The surgeon should use the method with which he is most comfortable.

The facial nerve should be preserved in the case of a benign tumor except in the patients with a recurrent, benign mixed tumor where the tumor has engulfed the nerve. In this instance, it is mandatory to excise the tumor even if the nerve must be sacrificed.

We feel that proper initial management of benign tumors of the superficial lobe is superficial parotidectomy. Small inadequate incisions lead to recurrences. On occasion, it is technically difficult to preserve the integrity of the nerve in benign tumors of the deep lobe. If the nerve must be transected, it should be anastomosed immediately after the tumor has been removed, if possible.

In our experience, frozen section diagnosis is very helpful, and we employ it routinely after the entire tumor has been removed. In those instances in which a malignant tumor is diagnosed, an attempt to preserve the facial nerve should be made if the malignancy can be thoroughly excised with an adequate margin of normal glandular tissue around it. However, in patients with highly malignant tumors such as squamous cell carcinoma, cylindroma, or malignant mixed tumor, the nerve is sacrificed if need be. It has not been our practice to re-establish continuity by nerve graft in these patients. While some authors[13] agree with this principle, others advocate immediate nerve grafts.[19]

As with other malignant tumors, the question of radical regional lymph node dissection must be considered. In patients where node involvement is present clinically, radical neck dissection is clearly indicated. Elective neck dissection is generally reserved by us and others for the highly malignant epidermoid carcinomas (mucoepidermoid, squamous cell)[6, 13, 14] or for those patients whose malignancies are highly invasive at the time of operation.[25]

Following adequate surgery, the prognosis varies according to the histological type of the tumor. In the largest series available, Frazell[13] reviewed 1549 cases of which 1267 were determinate. He found 98 per cent five year survival for all types of benign tumors of the parotid. Five year survival rates for malignant tumors were: malignant mixed, 55 per cent; mucoepidermoid—low grade, 96 per cent; high-grade, 14 per cent; squamous cell carcinoma, 36 per cent; adenoid cystic carcinoma, 40 per cent; acinic cell carcinoma, 82 per cent; adenocarcinoma, 48 per cent; cystic papillary lesion, 100 per cent; and unclassified malignant tumor, 24 per cent.

COMPLICATIONS

The major complications of surgery of parotid gland tumors are (1) recurrence, (2) facial nerve paresis, and (3) the Frey syndrome. Recurrence of benign tumors is practically confined to mixed tumors. Beahrs et al.[2] showed that this problem could be virtually eliminated by performing a superficial parotidectomy instead of enucleating the tumor. Once recurrent, these tumors are difficult to eradicate[25] and the incidence of facial nerve damage rises sharply in secondary resections.[4] Of the tumors classified by Beahrs et al.[2] as moderately malignant, an average recurrence rate of 51 per cent was recorded, while the average recurrence rate for lesions classified as highly malignant was 73 per cent. However, many of the tumors included in the series were not adequately treated by present standards.

While sacrifice of a portion or all of the facial nerve in the eradication of a malignancy may be difficult for the patient, inadvertent section of the facial nerve is a devastating experience in the patient with benign disease. It is difficult to obtain accurate statistics in the latter group of patients. State and Grage[28] found injury to a major branch of the facial nerve in 10 patients, and total division in 6 patients, in a series of 133 cases of pleomorphic adenomas. Eleven of these transections followed treatment of recurrent lesion. Hanna and Gaisford[17] purposely sacrificed the facial nerve in 17 of 300 patients. Brown et al.[7] reported no instances of facial nerve sacrifice in 115 patients with benign tumors. It has been our experience, and that of others, that meticulous superficial parotidectomy for benign lesions, with emphasis on identification of the facial nerve, should be rewarded with a minimum of permanent damage to the facial nerve.

The incidence of the Frey syndrome (auriculotemporal syndrome) varies with different reports because its presence or absence seems to depend upon how diligently its symptoms are sought. The condition is characterized by sweating and flushing of the face in the preauricular area after gustatory stimuli. This is caused by damage to the auriculotemporal nerve and it is thought that transected parasympathetic fibers grow along sympathetic pathways to the sweat glands. Its incidence has been reported in from 6 to 60 per cent[29] of patients who have undergone parotidectomy. However, fewer than 25 per cent of those who exhibit the syndrome are noticeably troubled by it.

SUMMARY

Tumors of the parotid gland are uncommon. In adults, one fourth are malignant; in children, one half of the solid tumors are malignant. The most common benign tumors are mixed tumors and Warthin's tumors, while the most common malignant tumors are mucoepidermoid tumors, malignant mixed tumors, and adenocarcinomas. The superficial lobe is more often affected than the deep. The tumor is usually found by the patient, and the physician confirms its presence.

Treatment of parotid tumors is surgical removal. In spite of adjunctive aids, accurate preoperative diagnosis is seldom possible. The patient and surgeon must be prepared for wide resection, which may include sacrifice of the facial nerve. Cervical lymphadenectomy is not done as a routine but only if there is clinical lymphadenopathy. Elective cervical node dissection is reserved for the highly malignant epidermoid, squamous cell, and high-grade mucoepidermoid carcinomas, and for those patients with highly invasive tumors at the time of operation. Cervical node resection is indicated for the remaining parotid malignancies only if there is good clinical evidence of metastasis.

The major complications of surgery on the parotid gland are facial palsy, the auriculotemporal syndrome, and recurrence of the tumor.

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Management of Cancer of the Parotid Gland

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A metropolitan community hospital attracting large numbers of cancer patients may reveal patterns of cancer presentation and development dissimilar to those seen in specialized cancer referral centers. The former is less apt to report statistics biased by substantial numbers of unusual or advanced tumors and is perhaps more representative of an average surgical experience.

In a 16 year period from 1955 to 1970, 70 patients with cancer of the parotid gland were registered in the cancer registry and treated at St. Francis Hospital. All were private patients, treated with uniform philosophy of management, and all patients treated are accounted for in this study.

Thirty-two patients were men and 38 were women. The ages ranged from 17 to 83 years, with peak and essentially equal incidences in the fifth, sixth, and seventh decades. Tumors were about equally distributed between right and left parotid glands. A striking familial incidence of cancer was noted: 23 patients (33 per cent) gave such a history. Histologically, this group of tumors was similar to those reported in other series:[2] mucoepidermoid, malignant mixed, adenoid cystic, and acinic cell carcinomas comprised three fourths of the entire group (Table 1). Substantially different survival rates at 5 years were noted for the various histologic types.

Metachronous cancer (Table 2) occurred in 7 patients (10 per cent) and was fatal in five. Only 1 patient developed a second cancer in the breast and she has remained well to date following mastectomy. Our group of patients is too small to permit comment regarding the alleged increased incidence of breast cancer in patients previously treated for parotid cancer.[1]

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Table 1. *Histologic Distribution and Survival in Carcinoma of the Parotid Gland*

HISTOLOGIC TYPE	NO. OF CASES	5 YR. SURVIVAL NED*
Mucoepidermoid	15	75%
Malignant mixed	11	36%
Adenoid cystic	13	42%
Acinic Cell	13	90%
Adenocarcinoma	5	25%
Squamous cell	8	25%
Poorly differentiated carcinoma	4	0%
Neurofibrosarcoma	1	100%

*NED = No evidence of disease. Percentages based on 63 patients followed 5 years or more.

Table 2. *Metachronous Cancers in Patients with Carcinoma of the Parotid Gland*

HISTOLOGIC PAROTID CANCER	METACHRONOUS CANCER	SURVIVAL	LAST STATUS
Malignant mixed	Alveolar cell* carcinoma, lung	252 months	Dead with disease
Malignant mixed	Carcinoma* of rectum	156 months	NED.** Died of other causes
Malignant mixed	Carcinoma of prostate*	144 months	NED. Died of other causes
Squamous cell carcinoma	Carcinoma of tonsil	45 months	Dead of disease
Squamous cell carcinoma	Carcinoma of breast	204 months	NED
Mucoepidermoid carcinoma	Bronchogenic* carcinoma	54 months	Died of other causes
Adenocarcinoma	Carcinoma of liver*	312 months	Dead with disease

*Fatal Cancer
**NED = No evidence of disease

Table 3. *Classification and Survival of Primary Patients*

	NO. OF CASES	5 YR. SURVIVAL NED*
No previous diagnosis or treatment	30	67%
Previous biopsy only	19	56%
Prior definitive treatment	21	20%
TOTALS	70	49%

*NED = No evidence of disease. Percentages based on 63 patients followed 5 years or more.

Table 4. *Primary Patients (4 of 30) with Preoperative Diagnosis of Parotid Cancer*

HISTOLOGIC TYPE	CLINICAL SIGN OR SYMPTOM	SURVIVAL
Malignant mixed	Palpable neck nodes	Dead of disease, 17 mos.
Malignant mixed	Ulcerated tumor mass	Dead of disease, 3 mos.
Adenocarcinoma	Facial nerve weakness	Dead of disease, 13 mos.
Poorly differentiated carcinoma	Palpable neck nodes	Dead of cerebrovascular accident, 18 mos. NED

Thirty patients (primary group) had had no previous surgical intervention. Nineteen patients had undergone only biopsy prior to referral, while 21 patients (secondary) had had prior definitive treatment and had been referred for treatment of recurrent parotid cancer. The survival rates for each group are listed in Table 3. Of the primary patients, only 4 were preoperatively thought to have cancer (Table 4) and, of these, only one demonstrated facial nerve paresis.

Throughout the period of this study, carcinoma of the parotid gland was considered primarily as a surgical disease. Table 5 lists types of treatment and results. No local excisions were performed. Patients treated by prior local excision were considered to have undergone biopsy only and were retreated by parotidectomy. The surgical technique of parotidectomy involved complete dissection of the facial nerve trunk and all its branches. No effort was made, however, to excise the "deep lobe" of the gland in those situations in which tumor appeared to lie entirely above the facial nerve. Radical parotidectomy (deliberate excision of the facial nerve) was employed when the nerve appeared involved by cancer or when adequate removal of a bulky tumor seemed compromised by efforts to spare the nerve. All patients classified in the secondary group were treated surgically although some received radiation therapy or chemotherapy as well. Although these patients had more advanced cancer than

Table 5. *Treatment and Survival in Cancer of the Parotid Gland*

TREATMENT	PRIMARY	PREVIOUS BIOPSY PRIMARY	SECONDARY	5 YR. SURVIVAL NED
Parotidectomy (nerve spared)	13	8	1	94%
Radical parotidectomy	9	4	7	55%
Radical neck dissection	7	4	1	36%
Palliation: Excision Radon seeds Cobalt Chemotherapy	1	3	12	0%

Table 6. *Survival of Patients Undergoing Secondary Treatment of Parotid Carcinoma*

TREATMENT	NO. OF PATIENTS	AVERAGE SURVIVAL IN MONTHS
Surgery	13	160
Surgery and radiation	7	120
Surgery, radiation, and chemotherapy	1	120

Table 7. *Radical Neck Dissection for Parotid Gland Carcinoma—Histology of Tumor and Node Status*

HISTOLOGY	NO. OF CASES	POSITIVE NODES
Malignant mixed	3	3
Squamous cell	4	0
Acinic cell	2	1
Adenocarcinoma	2	1
Adenoid cystic	1	0

Table 8. *Indications for Radical Neck Dissection in Carcinoma of Parotid Gland*

	NO. OF CASES
Histology of tumor	2
Prior biopsy or frozen section revealing positive nodes	3
Enlarged neck nodes clinically or at operation	7

Table 9. *Survival and Recurrence Rate in Patients Undergoing Radical Neck Dissection for Parotid Carcinoma*

	NO. OF CASES	5 YR. SURVIVAL NED*	NECK RECURRENCE
Nodes histologically positive	5	25%	60%
Nodes histologically negative	7	43%	57%
TOTAL	12	36%	58%

*Based on 11 patients followed more than 5 years.

Table 10. *Failure of Treatment and Status of Disease*

INITIAL PRESENTATION	LOCAL RECURRENCE OR REGIONAL METASTASES ONLY	DISTANT METASTASES ONLY	REGIONAL AND DISTANT METASTASES
Primary therapy	Living with disease – 1	Dead of disease – 4	Dead of disease – 3
Previous biopsy and primary therapy	Living with disease – 1	Dead of disease – 2	Dead of disease – 3
Secondary therapy	Living with disease – 1	Dead of disease – 4	Dead of disease – 5 Living with disease – 1

the primary or biopsy only groups, aggressive treatment appeared to be justified by the survival results (Table 6).

Radical neck dissection was employed in 12 patients for five different histologic types of cancer (Tables 7 and 8). The results of treatment for such patients according to node status is listed in Table 9. It is noteworthy that cancer recurrence in a previously dissected neck occurred with equal frequency in node positive and node negative patients.

Information on sites of recurrence was available in 25 patients. Treatment failure (Table 10) was overwhelmingly related to distant metastases and not simply to local or regional recurrence.

Eight patients survived unusual lengths of time despite locally recurrent or metastatic parotid cancer (Table 11). The majority had adenoid cystic carcinoma and here radiation therapy for local recurrence was occasionally of remarkable benefit.

Table 11. *Prolonged (>6 months) Survival in Parotid Cancer*

HISTOLOGY	TREATMENT	SURVIVAL	END RESULT
Adenoid cystic	Multiple excision, radical parotidectomy	240 months	Dead of disease, distant metastases
Adenoid cystic	Multiple excision, Radon seeds	192 months	Dead of disease, local and distant metastases
Adenoid cystic	Multiple excision, Cobalt	192 months	Dead of disease, cerebral metastases
Adenoid cystic	Multiple excision, radiation, chemotherapy	120 months	Living with disease
Adenoid cystic	Radical parotidectomy, chemotherapy	108 months	Living with disease
Malignant mixed	Multiple excision	250 months	Dead with disease, alveolar cell carcinoma, local recurrence
Malignant mixed	Multiple excision	240 months	Living with disease
Adenocarcinoma	Multiple excision, Radon seeds	310 months	Dead with disease, hepatoma

DISCUSSION

This group of patients with cancer of the parotid gland is probably representative of those patients most surgeons are asked to treat. Review of their disease suggests that adequate treatment of early parotid cancer is highly effective.

Two-thirds of previously untreated patients remained "cured" for 5 years. This high figure reflects the fact that treatment was instituted at early stages of disease, often when cancer was occult. Only 4 of 30 primary patients were preoperatively thought to have cancer; in our experience facial nerve weakness was an infrequent signpost of malignant parotid disease.

Preliminary excisional or incisional biopsy appears to affect unfavorably the prognosis of parotid cancer, though not to a great degree. Our figures suggest that the presence of a parotid cancer need not be (nor can it often be)[3] anticipated to affect a high rate of cure if sound principles of parotid surgery are exercised. Nevertheless, as a general rule (excepting perhaps with bulky lesions or suspected Warthin's tumor), we would discourage excisional or incisional biopsy of any lump considered to be a possible parotid neoplasm.

All patients receiving primary treatment with intent of cure were treated surgically. Our surgical philosophy was conservative, emphasizing preservation of facial nerve function. Results seem to amply justify this philosophy. However, since the majority of early parotid cancers are not suspected preoperatively, anything less than supraneural parotidectomy is inadequate. Frozen section analysis to identify the primary parotid tumor has not been sufficiently reliable in our experience to be used as a basis for determining treatment.

It is impossible to unequivocally assign the role of neck dissection in the management of parotid cancer.[4] We were startled to find that cancer recurrence rates were about equal in both histologically positive and histologically negative neck specimens. Furthermore, since nodes were positive in only 5 of 12 dissections, and three of these patients suffered recurrence in the dissected neck, the overall utility of neck dissection in the management of parotid cancer is open to question. We feel that patients treated with neck dissection should be selected with great discrimination and that the indications for neck dissection in parotid cancer be limited to histologically proven metastases. In this regard, frozen section on parotid or upper neck nodes may prove extremely worthwhile in making a decision for neck dissection. Additional efforts to control neck node recurrence are justified including preoperative radiation therapy to a neck where a prior biopsy has established the presence of metastatic cancer.

The presence of recurrent regional or distant metastases is not inconsistent with prolonged and useful life. Adenoid cystic carcinoma of the parotid gland, in particular, may run an extremely indolent course and when locally recurrent may respond dramatically to irradiation.

Although our best results were found in patients with acinic cell carcinoma, longer follow-up may substantially alter this impression as others have found a significant mortality from this disease after 5 years.

CONCLUSIONS

Cancer of the parotid gland is highly curable if properly treated in early stages of its development. Surgical treatment should consist of adequate removal of the tumor which is never less than superficial parotidectomy. Routine excision of the facial nerve is unnecessary and unjustified. The indications for neck dissection are few and perhaps should be limited to histologically proven neck node metastases.

A conservative surgical philosophy in selecting appropriate treatment is further justified by the fact that most treatment failures are related to distant metastases, not local recurrence. The natural history of parotid cancer is often favorable even in the presence of distant metastases. Inoperable local recurrence may occasionally be effectively treated by irradiation.

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Nasopharyngeal Cancer

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Nasopharyngeal cancer is a very common malignant disease of Chinese inhabitants of the Southeast Provinces of China. It occurs more commonly in males than in females. The age of highest incidence is 40 to 60 years. The cause of this disease remains unknown, although ethnic origin, environmental factors, and even virus infection may play an important etiologic role. The common presenting symptoms are cervical lymphadenopathy, nasal bleeding, hearing impairment, and cranial nerve palsy with diplopia and numbness of the face. More than 90 per cent of nasopharyngeal cancers are of epithelial origin, and more than half of them are undifferentiated carcinoma. Because of the technical difficulty of surgical treatment and the relatively favorable response to irradiation, the treatment of choice for the cancer of nasopharynx has been radiotherapy. The overall 5 year survival rate is around 30 per cent. Failures are mainly due to advanced local invasion to the base of the skull, brain, and other contiguous structures as well as to distant metastasis.

The table and figures appearing in this paper are from one of the authors' (Dr. Chiang) series of 350 proved cases of nasopharyngeal cancer treated at the Veterans General Hospital, Taipei, Taiwan, in the period from September 1961 to March 1970.

EPIDEMIOLOGY

The incidence of nasopharyngeal cancer is high among Chinese people living in the Southeast Provinces of China, namely, Kwangtung (Canton), Fukien, Kwangsi, and Taiwan. This malignant disease is also prevalent in Hong Kong, which is a part of Kwangtung Province. Biopsy data

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show that in the localities mentioned, nasopharyngeal cancer is the most frequent cancer in men and the third most frequent in women next to cancer of the uterine cervix and the breast. The highest incidence reported comes from data from the Kwangtung Province, where nasopharyngeal cancer constituted 56.9 per cent of 3010 malignant biopsies in men and 17.4 per cent of 4026 malignant biopsies in women. The incidence of this disease decreases fairly rapidly in the interior of China, and it is relatively rare in Northern China. At Peking, the percentage of nasopharyngeal cancer was 4 per cent of 5137 malignant biopsies in men.[3, 5, 15]

The relatively high incidence of nasopharyngeal cancer in the countries of Southeast Asia may be related to Chinese immigration. In Japan and Korea, this disease is very rare. In the Caucasian populations, the incidence of nasopharyngeal cancer is low and comprises about 0.25 per cent of all malignant tumors.[2]

Nasopharyngeal cancer in these regions also occurs more commonly in men than in women, with a ratio of about 2.5 to 1. In a series of 1438 cases in Hong Kong, the peak case incidence was in the 40 to 44 age group in both male and female patients.[8] This is about 10 to 15 years younger than the peak age incidence of patients reported in western countries.

ETIOLOGY

The high incidence of nasopharyngeal cancer among the Southeastern Chinese living either in their homeland or abroad suggests that these people may inherit a genetic trait that makes them more susceptible to this disease than other ethnic groups. However, no such genetic defect has been discovered, and no clinical data have revealed any preponderance of this disease among a patient's family or relatives. Of the Chinese living abroad, the locally born younger generations have a much lower incidence than the Chinese born older generations.[10, 16] This difference of incidence in the same race favors the "way of living" or environmental factor as being of etiologic significance, since if the genetic factor plays the only etiologic role, the place of birth would not affect the incidence. Studies of environmental factors such as cultural background, dietary habits, ventilation, and possible carcinogens in or around the house in which the patient has lived, and the frequency of drinking tea, using alcohol, and smoking cigarettes have been estimated to a certain extent in the countries in which this disease is prevalent, but none has been proved scientifically to be closely related to the cause of nasopharyngeal cancer.

The relationship of a virus and nasopharyngeal cancer has been studied recently.[7, 9] This study has shown that Chinese patients with nasopharyngeal carcinoma yield more evidence of infection of adenovirus types 7 and 12 than does the normal Chinese population, and an unusual frequency of high anti-Epstein-Barr virus titer in the serum as compared with the control group, who either have other head and neck cancers or

are relatively normal. For the Hong Kong patients, the serologic data were correlated with the disease-stage classification, and the incidence of high titer (1:160) showed successive increases from 45 per cent in early cases to 100 per cent in advanced cases. In patients in India and France, however, there was no increase in titer.

ANATOMY OF THE NASOPHARYNX

The anterior limit of the nasopharynx is the choana, which is continuous with the nasal cavities. Its roof is attached to the base of the skull and slopes downward and backward to become continuous with the posterior pharyngeal wall. Each of the two lateral walls contains the orifice of the eustachian tube, which is surrounded by the elevated ridge at its superior and posterior border, called torus tubarius. In between the lateral wall and the declining posterior wall lies an elongated depression called the pharyngeal recess, or Rosenmüller's fossa. The floor of the nasopharynx is the superior-posterior surface of the soft palate and the opening into the oropharynx (Fig. 1).

CLINICAL MANIFESTATIONS

The clinical symptoms and signs of nasopharyngeal carcinoma can be classified as follows.

The Presence of Tumor in the Nasopharynx

NASAL. Since most nasopharyngeal carcinomas are quite vascular and bleed easily, a blood stained nasal discharge is a common presenting symptom. When the tumor becomes larger, it may obstruct the nasal passage and the patient will tend to breathe through the mouth.

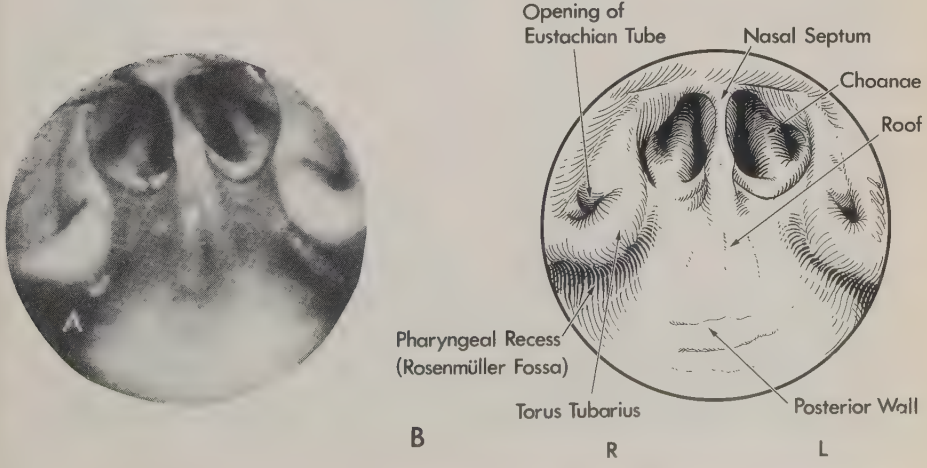

Figure 1. *A*, Normal nasopharynx by nasopharyngeal camera. *B*, Anatomical landmarks.

Table 1. *Common Presenting Symptoms of Nasopharyngeal Carcinoma*

COMMON SYMPTOMS	NO. OF PATIENTS	PER CENT OF 350 PATIENTS
Nasal bleeding	155	44.2
Nasal obstruction	106	30.3
Headache	141	40.2
Stuffiness, impaired hearing	79	22.6
Tinnitus	73	20.8
Diplopia, visual disturbance	70	20.0
Numbness of face	42	12.0
Sore throat	30	8.6
Hoarseness, voice change	18	5.3
Swallowing disturbance	15	4.3

OTIC. When the orifice of the eustachian tube is involved or completely occluded by the tumor, a variety of symptoms such as tinnitus, stuffiness, impaired hearing, and otitis media may occur (Table 1).

Metastasis to Cervical Lymph Nodes

The first echelon of lymphatic drainage of the nasopharynx is the parapharyngeal lymph nodes. Because these are difficult to examine, the

Figure 2. *A*, Bilateral upper cervical lymph node metastasis. *B*, The location and extent of the cervical lymph node most commonly involved by metastases.

Table 2. *Cervical Lymph Node Metastasis in Nasopharyngeal Carcinoma*

CLINICALLY POSITIVE	NO. OF PATIENTS		PER CENT OF 350 PATIENTS	
Unilateral	149		42.5	
Single node		90		25.7
Multiple nodes		59		16.8
Bilateral	138		39.5	
Single node		16		4.5
Multiple nodes		122		35.0
TOTAL	287		82.0	

actual incidence of involvement of this group of nodes is unknown. Nasopharyngeal carcinoma tends to metastasize to the cervical lymph nodes early. About 70 to 80 per cent of patients may show evidence of neck node metastasis when the diagnosis is made. The most commonly involved node is the upper deep cervical lymph node, which may be felt at the anterior margin of the sternocleidomastoid muscle, just below the ear lobe and posterior to the mandibular angle (Fig. 2). The jugulo-diagastric node is the next most common node found to be involved. The malignant cells then migrate and involve the spinal accessory chain, midjugular nodes, and other lymph nodes in the neck (Table 2).

Invasion of the Neighboring Structures of the Nasopharynx

THE CRANIAL NERVES AND BASE OF SKULL. Because of its anatomical proximity, nasopharyngeal carcinoma is notorious for its involvement of the cranial nerves and invasion to the base of the skull. The pharyngeal recess or the Rosenmüller fossa of the nasopharynx is almost always involved with the disease when the diagnosis is made. This recess, or fossa, provides a route for the cancer to advance to one of the foramina in the base of the skull, the foramen lacerum, which is the most frequently involved part of the base of the skull (Fig. 3). Lying immediately above the foramen lacerum is the cavernous sinus through which the third, fourth, fifth, and sixth cranial nerves pass to the rostrum. These nerves are therefore vulnerable to the invasive nasopharyngeal carcinoma. Of these four cranial nerves, the sixth nerve is the one lying lowest in the cavernous sinus and closest to the sagittal plane and therefore is the earliest cranial nerve to be affected: the patient complains of double vision and is found to have an inner squint because of paralysis of the lateral rectus muscle (Fig. 4). When the third and fourth cranial nerves are also involved, the patient presents with ptosis of the upper eyelid, and the eyeball is found to be fixed. The second and third branches of the fifth cranial nerve are the next involved and the patient usually complains of numbness of face and lips on the affected side.

Nasopharyngeal carcinoma may also advance lateroposteriorly from the pharyngeal recess into the retroparotidian space in which the ninth,

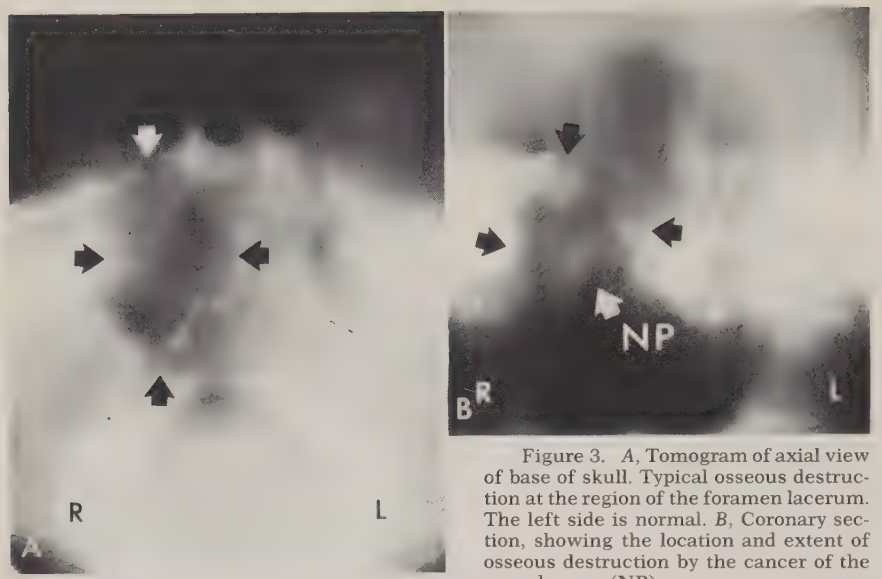

Figure 3. *A*, Tomogram of axial view of base of skull. Typical osseous destruction at the region of the foramen lacerum. The left side is normal. *B*, Coronary section, showing the location and extent of osseous destruction by the cancer of the nasopharynx (NP).

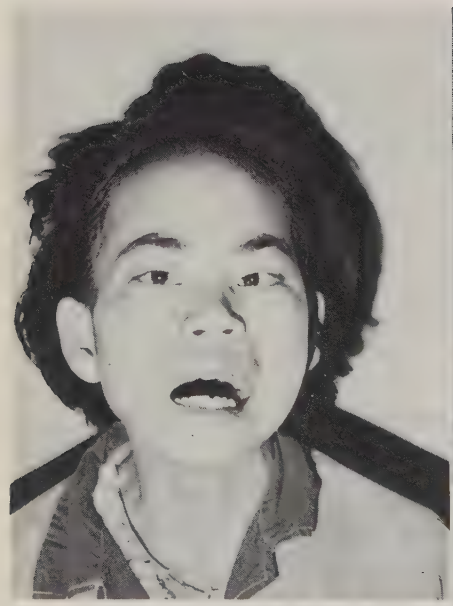

Fig. 4

Fig. 5

Figure 4. Diplopia and inner squint of the left eye with left sixth cranial nerve palsy. Trismus is also present.

Figure 5. Involvement of all but the first and second cranial nerves on the left.

tenth, eleventh, and twelfth cranial nerves run down vertically after they leave the cranium (Fig. 5). Also present in this region are the superior cervical sympathetic nerves. These nerves may be involved either directly with the local invasion or by the metastatic retropharyngeal lymph node. Patients with involvement of the ninth and tenth cranial nerves usually complain of hoarseness and trouble with swallowing. Upon examination, one may see characteristic signs pertaining to the impairment of the function of the involved nerve. Horner's syndrome is noticed when the sympathetic nerves are affected (Table 3).

The first and second cranial nerves are rarely involved because they lie rather far in front of and above the nasopharynx. The seventh and eighth cranial nerves are also less frequently affected because they are rather high and lateral in location and also are protected by the thick petrous bone. Another reason for the relative immunity of the eighth cranial nerve to this disease is its short course. Nasopharyngeal carcinoma may invade the forming bones of the base of the skull, the sphenoid sinus, and the meninges. When infection is introduced through the destroyed bony defect, meningitis may ensue.

INVASION OF OTHER CONTIGUOUS STRUCTURES OF THE NASOPHARYNX. When the tumor invades through the lateral wall of the

Table 3. Cranial Nerve Involvement in Nasopharyngeal Carcinoma

Total Number of Patients With Cranial Nerve Involvement	Percent of 350 Patients
89	25.4

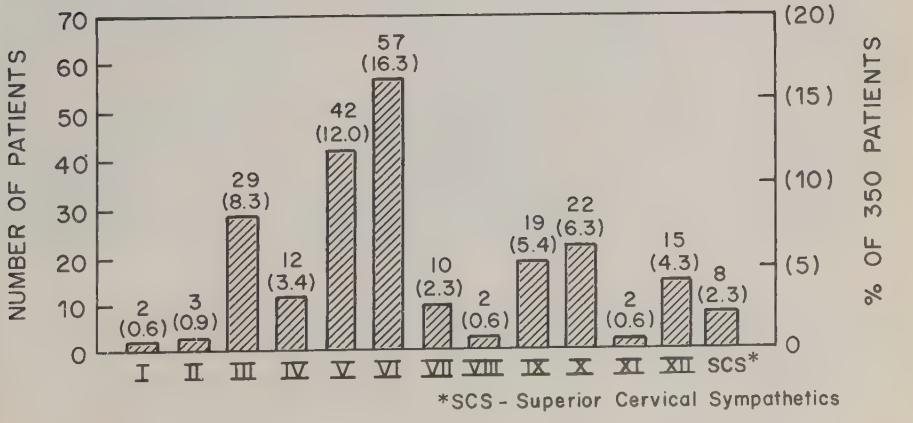

Table 4. *Local Invasion of the Contiguous Structures*

STRUCTURES	NO. OF PATIENTS	PER CENT OF 350 PATIENTS
Base of skull (including sphenoid sinus)	60	17.2
Oropharynx	28	8.0
Pterygoid fossa	15	4.3
Paranasal sinuses (excluding sphenoid)	7	2.0
Whole nasal fossa	5	1.4
Orbit	3	0.9

nasopharynx deeply into the pterygoid plate and muscle, trismus is a predominating symptom. When the retropharyngeal tissue and pharyngeal constrictor muscles are invaded, the patient usually complains of sore throat and difficulty in swallowing. The cancer may also grow and cause destruction of the posterior nasal structures, ethmoid, antrum, orbit, and oropharynx, producing a variety of clinical symptoms and signs in the organ involved (Table 4).

EXAMINATION

The patient with nasopharyngeal carcinoma deserves a thorough medical examination, with particular attention to the local invasiveness

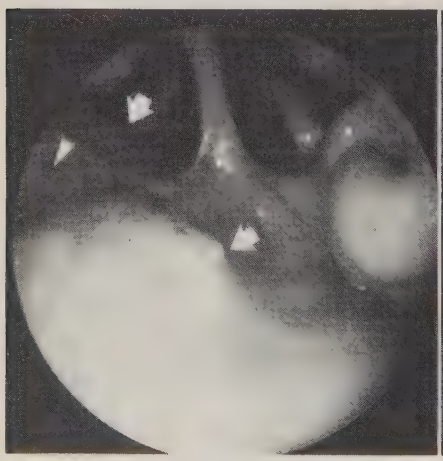

Fig. 6

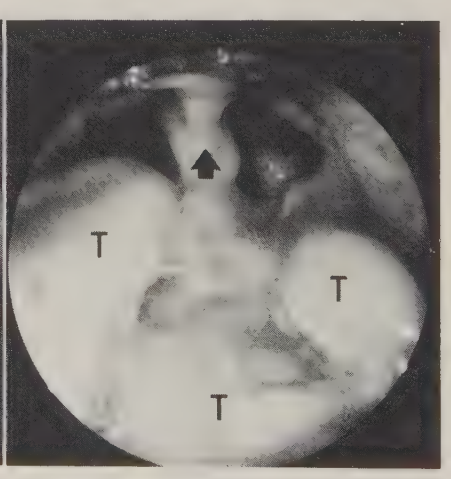

Fig. 7

Figure 6. The bulging tumor involves the right half of the nasopharynx. It obliterates the pharyngeal recess (*lower arrow*), occludes the orifice of the eustachian tube (*arrowhead*), and advances into the nasal fossa (*upper arrow*).

Figure 7. The tumor masses (T) involve the entire nasopharynx. The nasal septum is also infiltrated with cancer (*arrow*).

of the primary lesion, regional lymph node involvement, and signs of distant metastasis.

Mirror examination. Indirect mirror examination for the primary lesion in the nasopharynx can be performed without much difficulty by a skillful examiner. For a small percentage of patients who gag easily, topical anesthesia, 2 to 4 per cent lidocaine (Xylocaine) sprayed to the oropharynx, is helpful. On rare occasions, the patient may be so nervous that premedication with a sedative may be necessary. For patients with a short neck, a fat tongue, and a small nasopharyngeal cavity, an uvula retractor should be applied following topical anesthesia.

Nasopharyngeal camera. Topical anesthesia is applied to the oropharynx and an uvular retractor is applied. Through the oral approach, the whole nasopharyngeal cavity can be well visualized by the scope. The fiberglass optical system and camera are then attached and pictures can be taken for the desired locations. The advantages of this examination are manifold. It can demonstrate small lesions that cannot be detected by any other means. It provides a permanent record and is very useful on follow-up examination for residue or recurrent disease. Pictures taken before and after radiation therapy can always be put side by side for comparison and can serve as a guide in performing a biopsy so that many blind biopsies can be avoided (Figs. 6 and 7).

X-Ray examination. The presence of air in the nasopharyngeal cavity provides a natural contrast for demonstrating the tumor mass in the nasopharynx. The nasopharyngogram, which uses an opaque contrast medium to coat the wall of the nasopharyngeal cavity for better demonstration of the lesion, has been advocated by several authors. However, the primary function of the x-ray examination is for study of the bony structures that form the framework of the nasopharynx. Films of various projections should be obtained. Tomography proves to be most helpful for better visualization of bony destruction. The commonly involved bones are the sphenoid, the occipital, the tip of the petrous bone, the pterygoid plate, the paranasal sinuses, and the orbit.

DIAGNOSIS

Diagnosis of the nasopharyngeal cancer is based upon the presenting symptoms and the clinical findings of the patient. Nasal bleeding, enlargement of the upper cervical lymph nodes and double vision can be considered to be the triad signifying this disease, since when these three findings are present, the diagnosis of nasopharyngeal cancer is certain. The final diagnosis, however, should always be based on the histopathology of tissue taken from the nasopharynx.

PATHOLOGY

The mucous membrane that covers the nasopharynx is formed by a stratified cylindrical and ciliated epithelium.[1] The lymphoid tissue is abundant throughout the nasopharynx. The pharyngeal tonsil, or ade-

Table 5. *Pathological Classification of Nasopharyngeal Carcinoma*

TYPES	NO. OF PATIENTS	PER CENT OF 350 PATIENTS
Epidermoid carcinoma	151	43.1
Transitional cell carcinoma	133	38.0
Anaplastic carcinoma	37	10.6
Lymphoepithelioma	9	2.6
Angiosarcoma	1	0.3
Unclassified	19	5.4
TOTAL	350	100.0

noid, that is located in the superior-posterior wall, and the other lymphoid structures contribute to form the upper arch of the Waldeyer's ring.

Gross: The gross appearance of the nasopharyngeal carcinoma is most often of the exophytic type. It usually has a round smooth surface or is slightly lobulated. When the tumor arises from the posterior wall, its border may become less defined. The infiltrative lesion is infrequently seen, and the ulcerative type is extremely rare.

Microscopic: More than 90 per cent of cancers of the nasopharynx are of epithelial origin, either squamous cell carcinoma or, probably more commonly, the undifferentiated carcinoma, which may include the transitional cell carcinoma and lymphoepithelioma. The remaining less than 10 per cent may include lymphoma, adenocarcinoma, plasmocytoma, and a few other rare neoplasms.

Because of controversial opinions among pathologists on the interpretation and classification of the histopathological features of epithelial carcinoma of the nasopharynx, it is very confusing and almost impossible to compare the distribution of histological types of nasopharyngeal carcinoma from one series to another in the world literature. The lymphoepithelioma, originally described by Regaud and Schminche in 1921, which has been thought to represent one of the characteristic forms and has frequently been reported as a second most common type of nasopharyngeal carcinoma, has been the most confusing subject. It has been classified interchangeably with the transitional cell carcinoma by some pathologists, and frequently designated as undifferentiated carcinoma by others. A third group considers that the lymphoepithelioma should be discarded on the grounds that the lymphocytes appearing in the specimen merely represent accidental passengers from the abundant normal lymphoid tissue in the nasopharynx, and are not present in the metastatic foci[12, 13] (Table 5).

TREATMENT

The treatment of choice for nasopharyngeal carcinoma has been radiation therapy. The primary tumor is rather radiosensitive as compared

with carcinoma arising in other regions of the head and neck. The metastatic neck lymph node appears more so, and therefore radical neck node dissection is seldom necessary. Because of the anatomical location of the nasopharynx and the tendency of a cancer of this region to invade to the base of the skull and other neighboring structures, surgery at this location is almost impossible. No drug has been found to be effective in treating this disease. This may be due to the fact that few people have ever tried a well programmed course of chemotherapy for the disease at its advanced stage.

TECHNIQUE OF RADIOTHERAPY

Most hospitals are using megavoltage x-ray or cobalt-60 for treatment of nasopharyngeal carcinoma. These high energy photons are preferable to orthovoltage x-ray or high energy electron beams because they give a better depth dose and more homogeneity of isodose distribution in the heterogeneous medium of the nasopharynx, which contains air, soft tissue, and bone. Both the primary lesion in the nasopharynx and the neck nodes can be treated as a whole by using two lateral opposing portals. It is essential to include the base of the skull and both sides of the neck in the treatment field, even when there is no clinical evidence of involvement.

After 5000 rads in 5 weeks are administered to the midplane in this fashion, further irradiation of the neck is not advisable for patients who do not present with enlargement of the neck node initially. An additional 2000 rads in 2 weeks should be delivered to the primary lesion in the nasopharynx with a reduced portal size to avoid high dose to the brain or brain stem. This makes a total of 7000 rads in 7 weeks, an NSD of about 1950 rets, for the primary tumor. For patients with positive cervical nodes, more radiation to the neck is necessary and should be given by anterior and posterior opposing portals with a central shielding for the cervical spinal cord and larynx. The split-dose technique has been used by us since 1968 and seems to be more tolerable to the patients.

If the tumor recurs after a curative dose of radiation, it can be treated again with radiation either externally, or preferably by a combination of external x-ray beams and an intracavitary radioactive source.

Side Effects and Complications of Radiotherapy

Xerostomia. Unfortunately, the parotid and submandibular salivary glands must be included in the treatment field for nasopharyngeal carcinoma. After a curative dose of irradiation, almost all the patients will complain of dryness of mouth in the following years. To date, there is no effective way of protecting the salivary gland from the radiation injury.

Dental Defects. The dental defects acquired after irradiation develop whether the teeth are inside or outside the field of irradiation. They occur only when the salivary gland has been heavily irradiated.[6] The radiation-induced dental defect is similar to dental caries. Fluoride gel application to the teeth daily during and after radiation therapy proved helpful in reducing the incidence of caries formation.

RADIONECROSIS OF THE MANDIBLE. Since the introduction of megavoltage x-ray for clinical radiotherapy, the incidence of this complication following radiotherapy for head and neck cancer has been reduced greatly. However, the presence of dental caries, foci of infection in the gingiva or alveolar ridges, and periapical abscess within the irradiated field will predispose to the development of radionecrosis of the mandible, and therefore these conditions should be carefully evaluated by dentists before radiation therapy begins. When teeth need to be extracted, radiation therapy should not be given until the gingival tissue is healed. Radionecrosis of the mandible should be treated by surgery.

OTITIS MEDIA. This occurs when the primary lesion in the nasopharynx is locally advanced and the protection of the middle ear from direct beams of radiation is no longer possible.

CERVICAL RADIATION MYELITIS. When the radiation dose to the spinal cord is more than 6000 rads in 6 weeks, radiation myelitis may occur. It is usually seen 1 to 2 years following the radiation therapy. In a mild case, the patient may feel only tingling of the fingers. As the damage progresses, disturbance of both sensory and motor functions of the extremities develops, and the disease is frequently fatal in many of these patients.

PROGNOSIS

The prognosis of nasopharyngeal cancer depends upon the clinical stage of the disease, the adequacy of management, and the histopathological type. The average overall 5-year survival is about 30 per cent, with the highest reported series at 59 per cent using split-dose therapy.[11] The lymphoepithelioma may have a better prognosis than the well differentiated epidermoid carcinoma.[4] Failure to control the local disease of the nasopharynx occurs in patients who present with gross destruction of the base of the skull, multiple cranial nerve palsy, invasion to the pterygoid fossa, paranasal sinuses, and orbit. Metastasis to the cervical lymph nodes does not appear to be a grave prognostic sign. The distant metastases develop most commonly in bones, liver, and lungs,[13] and treatment for these patients is palliative.

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Cancer of the Floor of the Mouth and the Gingiva

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It has been estimated that deaths from oral cancer in the United States amounted to 71,000 cases in 1969, of which 53,000 were in men and 18,000 in women.[3] Thus, oral cancer was responsible for over 2 per cent of the total cancer mortality. Increased awareness, early diagnosis, and greater effort to achieve long-term control of the disease are strongly indicated. Primary importance in any program to reduce oral cancer mortality must be given to the management of *precancerous lesions.*

Carcinomas in the mouth have been reported to arise from ulcerative, traumatic, atrophic, inflammatory, luetic, and keratotic lesions of the epithelium. It is not possible to classify all lesions of the mouth as potentially malignant, despite the fact that reports of malignancy in all of these conditions have been claimed. Dyskeratosis is the most significant and distinctive histologic finding which signals the potential of malignant changes.[12] This is the sole epithelial alteration which consistently denotes the precancerous state.

Leukoplakia is the most frequently encountered dyskeratotic lesion of the oral cavity.[11] It is characteristically described as a grayish adherent patch. Clinically this description fits other common keratotic oral lesions such as focal-keratitis, nicotine stomatitis, and lichen planus. All of these lesions are either hyperkeratotic or dyskeratotic. The keratotic reaction is thought to be a mucosal defense mechanism against local irritants such as tobacco, chronic infection, or any thermal, mechanical, or chemical factor. True leukoplakia, or the dyskeratotic reaction, is a progression of the keratotic state into a definite premalignant lesion. Lichen planus is another lesion which must be differentiated from leukoplakia. Because of the difficulty in differentiating the disease clinically, particularly when

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Table 1. *Definition of the TNM Categories of Malignant Tumors About the Oral Cavity**

T	N	M
Primary Tumor	*Regional Lymph Nodes*	*Distant Metastasis*
T1S – Carcinoma *in situ*.	N0 – No clinically palpable cervical lymph node(s); or palpable node(s) but metastasis not suspected.	M0 – No distant metastasis.
T1 – Tumor 2 cm. or less in greatest diameter.	N1 – Clinically palpable homolateral cervical lymph node(s) that are not fixed; metastasis suspected.	M1 – Clinical and/or radiographic evidence of metastasis other than to cervical lymph nodes.
T2 – Tumor greater than 2 cm. but not greater than 4 cm. in greatest diameter.	N2 – Clinically palpable contralateral or bilateral cervical lymph node(s) that are not fixed; metastasis suspected.	
T3 – Tumor greater than 4 cm. in greatest diameter.	N3 – Clinically palpable lymph node(s) that are fixed; metastasis suspected.	

Stage I:	T1 N0 M0	
Stage II:	T2 N0 M0	
Stage III:	T3 N0 M0	
	T1 N1 M0	
	T2 N1 M0	
	T3 N1 M0	
Stage IV:	T1 N2 M0	T1 N3 M0
	T2 N2 M0	T2 N3 M0
	T3 N2 M0	T3 N3 M0
	Or, any T or N category with M1.	

*American Cancer Society. Cancer Statistics, 1969.

the lichen planus is hypertrophic, biopsy becomes mandatory to histologically differentiate between them.

Leukoplakia is most commonly found in the fourth through the sixth decades of life, more commonly in men than in women. The cause is unknown, and hence preventive measures must be directed at the controllable mechanism – the irritating factor. Most frequently condemned as irritants are tobacco, hot or highly seasoned foods, and mechanical factors such as ill-fitting dentures.

It is questionable whether leukoplakia is a reversible lesion. If the known irritating factor can be removed, treatment may be limited to observation, anticipating improvement in appearance or complete clearing of the lesion. Should neither of these obtain, excision of the lesion and histologic examination is mandatory. It is particularly advisable to remove lesions amenable to complete local excision before they attain such

a size that tissue replacement for coverage would be necessary upon removal.

It is common for leukoplakia to recur at the site of the excision, especially if the irritating factors such as smoking are not completely removed. Even when all obvious irritants have been removed, leukoplakic patches can reappear in the area of previous excision. This is a firm indication for re-excision. Other modalities for treatment of leukoplakia have been suggested, including any protein destructive agent such as topical acid or chemotherapeutic agents. Dermabrasion or split-thickness excision has likewise been described, and the use of wire abrading brushes by the patient, over a period of time, has been recommended. On thick mucosa, such as that of palate or gingiva, split-thickness excision has been successful in our experience. On the floor of the mouth surgical excision of the mucosa in full-thickness remains our treatment of choice. In extensive areas of involvement, the use of skin grafts for replacement of the oral mucosa is recommended,[11] despite the reluctance of some surgeons to resort to extensive procedures in the absence of proven malignancy of the lesion.

Systemic chemotherapy has also been used in the treatment of leukoplakia of the mouth. Methotrexate has been the agent most commonly used and has resulted, when given in adequate doses, in some resolution of these lesions. However, there has been a high incidence of recurrence of the lesions when chemotherapy was withdrawn.

Clinical staging of tumors of the oral cavity is desirable and the staging system of the American Cancer Society, shown in Table 1, is that which we follow.[3] Staging is based on the anatomic extent of the disease prior to first definitive therapy, and the stage is not changed thereafter. It is based on careful clinical examination, and proper staging is essential to choice of therapy and accuracy of statistical follow-up.

CARCINOMA OF THE FLOOR OF THE MOUTH

Squamous cell carcinoma of the floor of the mouth is the most common oral malignancy and comprises approximately 40 per cent of all oral cancers seen in our patients. This includes lesions of the underside of the tongue. They occur in a male to female ratio of about 20 to 1. It is a disease of the aged.[8]

Carcinoma of the oral cavity, particularly in patients who have leukoplakia, is often multifocal in origin and the patient who has had one carcinoma in the oral cavity has a significantly increased chance of developing future primary carcinomas.

In carcinoma of the floor of the mouth, it is interesting to distinguish the tobacco chewer's lesion (histologically described as verrucous carcinoma) from the usual squamous cell carcinoma. Verrucous carcinoma usually occurs on the buccal mucosa of the lower gingiva or anterior floor of the mouth and initially appears as leukoplakia. Gradually the lesion changes to a large exophytic papillary growth which may become infected and slowly invade the contiguous structures. Metastasis from this type of tumor is less common than from the invasive squamous cell

lesion. Histologic sections show swollen retepegs, extending into the deeper tissues. Local resection is the treatment of choice unless there is evidence of metastatic involvement of lymph nodes, in which case a combined operation,[13] consisting of resection of the primary oral cavity lesion with a regional node dissection in continuity, is the recommended treatment.

The common lesion of the floor of the mouth is the ulcerating invasive squamous cell carcinoma. This lesion may start near an area of leukoplakia or as a carcinoma in situ. It gradually forms firm indurated borders and often does not show great differentiation. Metastasis is primarily through the lymphatics.

Carcinoma of the anterior floor of the mouth should be considered separately because of the specific anatomic facts. The geniohyoid and mylohyoid muscles are directly inferior to the mucosa of the floor of the mouth and are considered a barrier between the oral cavity and the neck. At the posterior border of the mylohyoid, however, there is direct communication with the submaxillary compartment of the upper neck. Involvement of this posterior area precludes the therapeutic choice of *local* resection. Excision in continuity with neck structures is indicated. Neck metastasis occurs by direct or lymphatic dissemination or a combination of both. Lesions of the anterior floor of the mouth often involve the orifice or the submaxillary duct, which enters the mouth adjacent to the midline and may act as a conduit for tumor extension to the submaxillary triangle.

Midline lesions of the mandible or the anterior floor of the mouth present difficulties in treatment because of their location, relative to lymphatic drainage. It has been the practice of the authors to treat the patient with lesions which cross the midline with bilateral supra-omohyoid neck dissection. If there are palpable nodes in either side of the neck, a complete neck dissection is done on the side of the lymphadenopathy. When a supra-omohyoid neck dissection specimen is removed, it is carefully examined grossly. Lymph nodes are examined by frozen section. If histologic evidence of tumor in nodes is demonstrated, a complete neck dissection on the side with nodal metastasis is done. However, one must be aware of the fact that metastases to Levels III and IV in the neck may occur without nodal involvement at Levels I and II.[8] Therefore, careful follow-up is essential.

1940 marked the high point in the use of irradiation for cancer of the floor of the mouth.[8] Thereafter and gradually it became commonly accepted practice to utilize the surgical approach to lesions of the *anterior* floor of the mouth because of the better anatomic access to this area for resection in continuity. In contrast, lesions of the *posterior* floor of the mouth were usually irradiated because of the supposed difficulty of anatomic approach. With the development of en bloc surgical techniques, however, technical access to both areas was shown to be feasible, and the surgical approach to oral cavity tumors with in continuity resection of the neck structures is the recommended therapeutic choice.[5, 8]

The postoperative problems after resection of an anterior segment of the mandible are loss of support of the hyoid, with consequent difficulty in swallowing, and loss of sensation in the lower lip, which contributes to

drooling. In resections of posterior segments of the mandible, hyoid support remains intact and some sensory innervation is obtained from the contralateral side of the lip; thus rehabilitation of the patient is facilitated. In addition, preservation of the tip of the tongue and the contiguous structures in posterior resections, as opposed to anterior, results in a lesser degree of speech impairment. Posterior resections do not produce an Andy Gump deformity. Flattening of the side of the face with loss of a lateral segment of the mandible is more acceptable cosmetically than loss of the chin.

Conservative local resections are selected on the basis of the following criteria: (1) The primary lesion is clinically mobile and not invading the periosteum. (2) There is no X-ray evidence of bony invasion. (3) There is no suspicion of cervical node involvement. (4) The lesion by examination clinically is anterior to the free border of the mylohyoid muscle.

Indications for neck dissection have come under critical review in recent years.[10] Crile[7] states that carcinoma of the head and neck kills by local extension of the disease, or by extension into the neck nodes. Most patients who die from carcinoma of the head and neck have disease involvement limited to the area above the clavicle. Contraindications to neck dissection except for palliation are distant metastasis beyond the neck and direct extension to the base of the skull or cervical spine. Great latitude is recommended in decision for surgical exploration, since nonintervention usually results in a prolonged, painful waiting period with eventual death.

Indications for a neck dissection in cases of carcinoma of the floor of the mouth[10] are (1) palpable neck nodes; (2) the presence of neck metastases before irradiation; (3) failure of control of the primary lesion by irradiation; (4) primary lesion greater than 3 cm.;[8] (5) presence of a primary lesion whose excision required a surgical approach that entered the neck.

Those who opt for prophylactic neck dissection claim that 25 to 40 per cent of the specimens show micrometastatic disease in the nodes, particularly if the primary lesion is in the tongue, larynx, or another area with a high rate of metastasis. There is general agreement, however, that prophylactic neck dissection is indicated when the primary lesion is directly continuous with the neck, as previously noted with the floor of the mouth lesion extending posteriorly to the free border of the mylohyoid.

Anatomic metastasis from the floor of the mouth follows, in general, the lymphatic vessels in continuity with the plexuses of the tongue on one side and the gingiva on the other.[18] Anteriorly, the vessels pierce the mylohyoid muscle and pass to the submental lymph nodes (two or more on either side of the midline). The disease then progresses:(a) to the submaxillary lymph nodes, and (b) to the deep cervical nodes situated on the superficial surface of the internal jugular vein. Lesions adjacent to the midline of the floor of the mouth metastasizing or extending to the submental region (rather than to the submaxillary triangle) may require sacrifice of all of the floor of the mouth muscles – the mylohyoid, geniohyoid, and genioglossus. Seventy-five per cent of lesions of the floor of the mouth have invaded one or more of the adjacent structures by the time the diagnosis is first made.[8]

Local lymphatics are not believed to enter the mandible from the floor of the mouth. They tend to pass either posteriorly or laterally to the periosteal lymphatics.

Surgical access to the anterior floor of the mouth is obtained transorally or through the midline lip-splitting incision.[5] For extensive resections or combined resection, access is best obtained through a lip-splitting incision combined with the incision for a neck dissection. Variations have been described, such as the pull-through operation in which primary resection of the floor of the mouth and in continuity neck dissection are accomplished without removal of a segment of the mandible. This procedure is done by direct pull-through, or by splitting the mandible from the oral to the submandibular incision and re-wiring it. Limited access procedures are being utilized with lesser frequency in our personal experience.

Tumors which are amenable to successful treatment by extirpative surgical measures are often also amenable to treatment by radiation, and vice versa. Experience shows that the malignant tumor which does not respond to treatment in one manner does not often respond to control by any other method. This is probably due to the inherent virulence of the lesion. The morbidity of bone radiation influences our preference for therapy with surgical excision[8, 19] Successful management of the tumor by radiation (including bone) with subsequent exposure of the bone for any reason often requires resection of a much wider segment of bone than primary surgical excision.

Results of treatment of carcinoma of the floor of the mouth reported by Harrold over a 29 year period in determinate patients was 36 per cent 5 year survival (28 per cent of 804) with more than one hundred failures in this group representing patients who received either palliation or no treatment.

Five year survival, by stage, reported by the American Joint Committee for Cancer Staging and End Results Reporting February, 1967, was as follows:

	N0		N1	N2	N3
T1 stage 1	21/31	68%	2/2	0/1	0/5
T2 stage 2	21/29	70%	12/23	1/5	2/8
T3	4/9		5/12	0/4	0/10
	stage 3 = 50%		stage 4 = 9%		

Reconstructive Techniques in Excising Floor of the Mouth Cancers

Tissues removed from the floor of the mouth for purposes of control of malignancy should be restored for purposes of function. Excision of small lesions of the floor of the mouth allows some leeway in permitting primary closure without reconstructive procedures. It is mandatory to restore oral cavity lining to obtain proper function in cases in which primary closure necessitates binding the tongue to the cheek or lip. Mucosal reconstruction can be obtained in several ways. Popularity of various pedicle flaps has diverted attention from the simple and time honored use of the split-thickness skin graft for immediate coverage of oral defects following surgical procedures for carcinoma of the floor of the mouth. In post-irradiation cases, pedicle flap coverage is mandatory.[6]

Thin grafts have a greater chance of survival than thick ones, although the latter show less contracture postoperatively. Split-thickness skin grafts in the oral cavity must be stented into place. Balls of cotton or waste dressing, wrapped in various non-adhering dressings, have been used as well as dental compound, rubber tubing, molds, prostheses and other devices. Despite contamination by oral fluids, a high percentage of successful grafting has been achieved.[6] The disadvantages of the split graft are contracture and occasional failure of the graft. The advantages of split-thickness skin grafts include immediate availability. Also, a thin skin graft (as opposed to a bulky flap) may permit earlier observation of persistent tumor.

The use of pedicle flaps in restoration of oral mucosal integrity was popularized by McGregor who used the forehead flap for this purpose.[15] This flap has a vigorous blood supply which allows the use of the entire forehead skin (based on temporal vessels) to be turned into the mouth without previous delay. The flap may be turned through the cheek, below the zygoma, and reach the contralateral portion of the oral cavity. The primary objection to the use of the forehead flap is the donor site scar. With a split-thickness skin graft covering the forehead obtained from the lower neck or upper chest, resultant deformities are minimized because of the excellent match of the texture of the skin.[19] It is better to remove the entire forehead flap rather than replace part of it, particularly in men, because of the aesthetic deformity common with partial use of the forehead.

For reconstruction of the anterior floor of the mouth, the medial based deltopectoral skin flap, described by Bakamjian to reconstruct the pharynx, has now proven itself and is readily available.[1] Bakamjian has advised flap delay if feasible. In our experience, it is not necessary to delay this flap, if care is taken to preserve the perforating vessels from the internal mammary, at the medial base of the flap. The only exception to this rule, which would necessitate delay, has been extension of the flap beyond the shoulder region or marked kinking intra-orally. The usage of the deltopectoral flap is without doubt one of the most dramatic and useful surgical procedures which has been made available in recent years and has markedly expanded the frontiers of surgery of the head and neck.

In cases of small mucosal defects of the oral cavity, where the deltopectoral or forehead flap is not required, a nasolabial flap is available to resurface the floor of the mouth.[19] The usage of bilateral nasolabial flaps can adequately resurface the floor of the mouth and give considerable mobility to the tongue. These flaps are based inferiorly and tunneled into the mouth through the cheek by blunt dissection. After division of the pedicle, the tunnel is closed and the area of the base is revised as a secondary procedure.

The use of posterior neck flaps, thoracoabdominal tubes, pedicles, and shoulder flaps based laterally have largely been abandoned, because of the versatility and potential length of the deltopectoral flap described by Bakamjian. It is important, however, to keep these other flaps in the armamentarium to be utilized in the event of previous surgical scarring

or radiation changes in the area of the sternum, thus interdicting usage of the deltopectoral flap.

The usual defects requiring reconstruction following oral carcinoma surgery are: (1) deficiencies in external covering, such as the cheek or the lip; (2) deficiencies of mucosa and exposure of cavity, such as of the antrum or nose; (3) limitation of motion, such as binding down of the tongue by primary closure, or of limiting mandibular opening. At present, it is disheartening to read of surgeons resecting a mandible, not for purposes of gaining additional margin in excision of malignancies, but for purposes of closing the wound as previously described.[19]

TREATMENT OF CARCINOMA OF THE GINGIVA AND ALVEOLAR PROCESS

Cancer of the gingiva is representative of the general category of intraoral epidermoid carcinoma.[2] Significant gains have been made in the control of this cancer, which may occur in any part of the upper or lower jaws. Treatment of small malignant tumors consists of wide, local excision. Treatment of larger tumors (over 2.5 cm.), or those which have metastasized, requires composite operations, resecting the primary tumor, the contiguous structures, and the area of lymphatic drainage, and utilizing neck dissection. The treatment of carcinoma of the gingiva is essentially a surgical problem. Irradiation therapy is limited by the high incidence of post-irradiation complications of the underlying bone.

Innumerable techniques have been described for resection of lesions of the alveolar ridges. These include both partial-thickness and full-thickness resections of the mandible in conjunction with its contiguous structures, which may include portions of the floor of the mouth, cheek, and neck. Partial resections of the mandible include resection of the inner cortex, the alveolar ridge, or a combination of both, depending on the area of the primary tumor. Resection of the alveolar ridge is known as a marginal resection of the mandible. The purpose of these surgical procedures is to leave intact the mandibular continuity and avoid the postoperative difficulties which accompany loss of this continuity. In the patient in whom resection of the full thickness of the mandible with loss of continuity is indicated, immediate or delayed reconstructive efforts are recommended. These include bone grafting; stabilization with plates, pins, rods, or other inert material; and the usage of dental splinting and intermaxillary fixation to prevent drifting. If the lateral portion of the body of the mandible must be resected and the anterior body continuity is maintained, minimal deformity results.

The Andy Gump deformity results if there is no reconstruction of the mandible. When a segment of the mandible is resected, both lateral portions of the body of the mandible tend to drift inward, with the tongue falling into the mouth. In addition, hyoid support is lost and swallowing becomes difficult. One of the most disturbing problems is drooling, which is seen in the patient who has lost the anterior part of the mandible with

the sensory nerves to the lower lip. The use of prosthetic reconstruction of the anterior mandible at the time of the primary surgical resection has been discredited over a period of years because of oral contamination of the wound with resultant dislodging or erosion of the underlying prostheses through the mucosa or skin. It must be borne in mind, however, that despite eventual loss of these devices, the use of antibiotics and oral hygiene often permits them to be retained for 6 weeks or longer. Contraction of the wound is then less and alignment of the residual lateral portions of the mandible is maintained until definitive therapy by bone grafting may be done.

Free bone grafts are most useful in cases in which the resection is confined to the horizontal ramus of the mandible on one side with a relatively small gap and in the presence of adequate remaining mucous membrane or skin coverage. Sources of the bone graft may be either the iliac crest or the rib. The bone block is shaped to fit into the bony gap; one surface of the graft is cancellous, the other is cortical bone. The cortical bone will give a structural strength to the bone graft and the cancellous bone will facilitate capillary in-growth into the graft. Immobilization by maxillary wiring or other means for a period of 4 to 6 weeks is necessary for graft take.

The difficulty of preventing oral contamination of the bone graft makes us recommend the conservative approach to reconstruction of the mandible,[19] that is: (1) Restoring oral cavity lining. (2) Maintaining mandibular occlusion with the maxilla through the use of dental splints or interdental wiring or prostheses. Subsequently, after healing of the soft tissue wounds, mandibular continuity is restored with an autogenous bone graft.

Primary autogenous bone graft is the procedure of choice for reconstruction of the mandible. The desire on the part of surgeons to minimize morbidity has been responsible for the development of more and more complicated and larger procedures devised to correct the entire condition at the time of the primary extirpative surgery. Millard[16, 17] has demonstrated bone grafting and restoration of the mandible which can be performed at the time of the original resection with the use of a forehead flap for lining and a free graft from the rib. The popularization of the deltopectoral medially based flap, with its excellent blood supply, has extended this principle so that immediate flaps used for coverage of primary bone grafts in replacement of resections of the anterior floor of the mouth have been attempted more frequently. We would hope that this will be the path in the future as, without question, every effort should be made to reconstruct the oral cancer patient and to rehabilitate him as quickly as possible. These latter techniques are feasible now in the cases in which mucosal integrity in the mouth is maintained to prevent contamination by oral fluids. Unfortunately, most of the lesions which we have to treat involve mucosa and do involve contamination by oral fluids. The need for secondary reconstructive techniques therefore remains, because of the limited success in primary reconstruction techniques. We feel that restoration of mucosal lining to minimize the defect and secondary bone grafting is still most often the procedure of choice.

SUMMARY

There has been an improvement in survival rates of patients with cancer of the floor of the mouth since World War II and an even more significant improvement with cancer of the gingiva. These increased survival rates have been accomplished primarily with surgical treatment.

Surgical techniques have been developed which lead to less deformity and improved postoperative function.[2] With the increased use of surgery for patients with intraoral carcinomas, the demands on reconstructive surgery have correspondingly increased. Surgeons have become aware of the fact that there are virtually few limits on extirpative surgery of the head and neck, these being the spine, the base of the skull and one carotid; all other structures may be sacrificed if need be. Thus again the demands on the reconstructive surgeon have increased as more radical surgical procedures for control of carcinoma are attempted. Palliation for fungating, draining, or painful lesions may necessitate even more extensive procedures.

Formerly, there was a great reluctance to do surgical procedures that required large pedicle coverage, as this usually meant months or years of hospitalization with correspondingly multiple surgical procedures. Large defects around the face, jaws, and mouth required construction of a thoracoabdominal or other pedicle flap with multiple delays and transfer procedures and questionable success at the end of the long road.

Fortunately, reconstructive surgery responded to the challenge permitting us now to resurface and fill large head and neck defects from several donor sites without delay at the time of the primary extirpative surgery, if necessary. This additionally permits the surgeon responsible for resecting the tumor, be he the same or another surgeon, to treat the tumor as indicated without regard or worry for closure. This can now be done almost without exception.

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Cancer of the Tongue

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The management of head and neck cancer presents a unique opportunity for the cooperative efforts of multiple disciplines in order to achieve both maximum cure rates and maximum rehabilitation. It is particularly gratifying to those working frequently in this area to note the evidence of cooperation now so frequently seen. While a healthy competitive spirit continues to exist between surgeons and radiotherapists, the present recognition and acceptance of the other's potential contribution is more in evidence today than any time in the past. The expertise of many disciplines in addition to the two traditional competitors must be drawn upon to achieve optimal total rehabilitation of the patient. As these interrelationships are developed, it is apparent that only through careful advance planning can the contributions of the various specialties be employed at the time that it will be most effective and not merely convenient.

Over 95 per cent of the tongue cancer is of the squamous cell type, and the discussion will be confined to this lesion.[3] The tongue tumors present two different types of problems, depending on the anatomic area of the organ where the cancer develops. Tumors of the mobile tongue or anterior two thirds have a distinctly more favorable prognosis than tumors of the posterior third and base. Similarly, the management, particularly of the T_1 and T_2 lesions, is generally much less complicated.

SURGICAL TREATMENT

Surgical excision is the treatment of choice for the small superficial lesions of the mobile tongue (Fig. 1). A majority of these are on the lateral border and are readily accessible to partial glossectomy and tongue reconstruction. Even somewhat larger but very superficial lesions accompanied by areas of leukoplakia can also be handled as a transoral partial glossectomy (Fig. 2). A skin graft may be quite useful in these circumstances if a large portion of the mucosal surface must be sacrificed (Fig.

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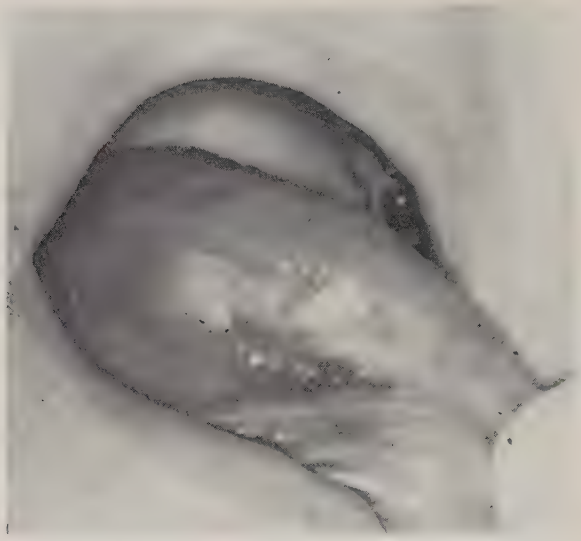

Figure 1. A superficial cancer at the juncture of the anterior and middle thirds of the tongue, accompanied by leukoplakia. Simple excision and primary reconstruction were undertaken.

Figure 2. Multicentric but superficial squamous cell cancer over the anterior two thirds of the tongue along the lateral border but extending also onto the dorsal and undersurface. Surgical excision was performed.

Figure 3. The sizable loss of mucosal surface following the removal of the tumor in Figure 2 required a split thickness skin graft to cover the defect. This is held in place with a series of interrupted sutures tied over a nylon and gauze stent. The stent is removed in 72 hours.

3), as the functional end result will be superior even though there is some shrinking of the graft (Fig. 4). Cady[4] has recently described an electrocoagulation technique, originally developed by Franseen, that leaves a large granulating surface. He feels, however, that this is superior in that it allows a careful observation of the local area for recurrence. The technique is somewhat analogous to the chemosurgery of Mohs in that the tumor is removed in a series of layers by a form of coagulation. There is no microscopic control of the specimen with electrocoagulation as there is with chemosurgery competently performed. Reliance for depth of excision is placed on the gross appearance of the coagulated tissue. Antibiotics are administered postoperatively when there has been intraoral reconstruction following partial glossectomy, though they are unnecessary if the wound has been left to granulate.

Larger lesions (T_3) often will require opening the neck in order to acquire satisfactory exposure of the primary tumor (Fig. 5). Whenever this is necessary in the treatment of cancer of the tongue, a neck dissection is combined with the removal of the intraoral disease. Whenever there is clinical evidence of metastatic disease in the neck, a neck dissection is performed and, if anatomically reasonable, it is done in continuity with the treatment of the intraoral primary. This is occasionally impractical, particularly when dealing with a small lesion of the anterior half of the tongue.

Cancers of the posterior third and the base of the tongue are generally treated by radiation (Fig. 6). Again, however, if there is clinical evi-

Figure 4. The same patient in Figures 2 and 3, one year after operation. There has been considerable shrinkage of the graft but its functional and cosmetic result is still superior to primary closure.

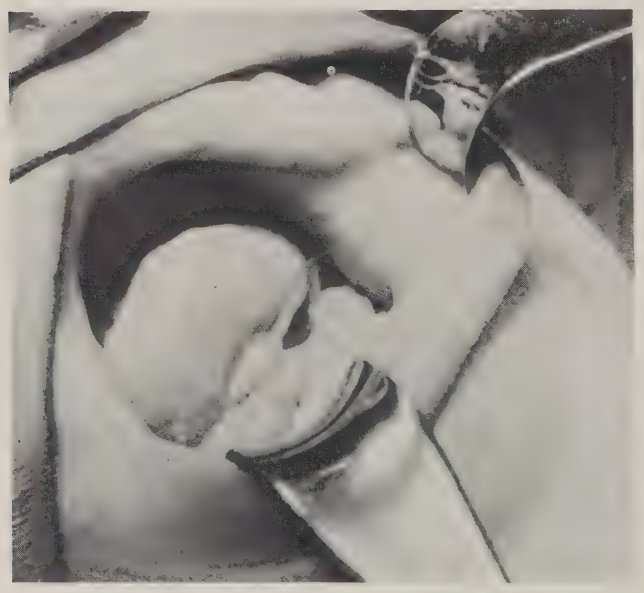

Figure 5. A small ulcer on the undersurface of the tongue near the border with the floor of the mouth belies a deeply infiltrating lesion. There was also clinical evidence of metastatic disease in the neck. The patient was treated with a Monobloc procedure following preoperative irradiation.

Figure 6. A large infiltrating squamous cell cancer of the posterior third and base of the tongue. The lesion extended across the midline. This was treated primarily with radiation, although 3 months postoperatively residual disease was noted. A Monobloc procedure was then performed. The patient shows no evidence of recurrent disease more than a year after operation.

dence of metastatic disease in the neck, a neck dissection should be performed. It generally can be done in continuity with the removal of the intraoral tumor in favorable circumstances. For some patients with relatively small tumors at the base of the tongue without evidence of metastatic disease in the neck, Tollefson and Spiro[28] have suggested the median labiomandibular glossotomy approach that they have now used on 13 patients. This would seem to be a useful exposure for a small highly selected group of patients for whom radiation was contraindicated.

Monobloc Procedure

For the sake of simplicity, a radical neck dissection combined with the removal of an intraoral tumor in continuity, with or without removal of a portion of the mandible, is referred to as a monobloc procedure. Careful planning of the entire operation by the multiple disciplines that may be involved before the patient is rehabilitated is essential to the overall success of the undertaking.[4] The patient must be thoroughly apprised of the exact nature of his problem and specifically what he can expect in the way of residual functional and cosmetic deformity as well as rehabilitation. His general physical status should be thoroughly evaluated, particularly the hepatic and cardiopulmonary function. Care should be directed toward local oral hygiene preoperatively, although this is not quite as important in these circumstances as it is in a patient who is to be subjected to a full course of radiation therapy. If the patient is a cigarette smoker, and a high percentage of the patients are, this should be discon-

tinued if only for a few days preoperatively in an effort to diminish the postoperative pulmonary complications.

We and others are not as yet certain of the value of the preoperative treatment of the primary tumor with radiation.[27] However, we do share the observation that there is a definite decrease in the incidence of local recurrence in the neck following a course of preoperative therapy. The short course which we now employ consists of roughly 2000 rads given in four treatments over the course of 4 or 5 days. This rarely delays the operative procedure, as the other preoperative preparation is being undertaken at this time. We have noticed only very rare major postoperative complications associated with this dose schedule. However when the preoperative treatment is given even with fewer rads in only one or two treatments the complication rate does rise significantly.[13, 29]

When there is to be a significant soft tissue or bony loss from the operative procedure, that individual who will be responsible for the reconstruction should participate in the preoperative planning. Something as simple as the positioning of incisions may make a considerable difference in the ultimate development of pedicle flaps and functional reconstruction.

Our success with primary bony reconstruction has been unsatisfactory. Similarly we have found that while heavy prosthetics are poorly tolerated, a simple Webb bolt (Fig. 7) or occasionally a Kirschner wire can be left in place and will maintain the stability of the bony fragments quite satisfactorily until the osseous reconstruction can be done in a sterile field. We feel strongly that functional rehabilitation is of the utmost importance and that closures of orocutaneous fistulas should be undertaken immediately or at least as soon as practical, considering the tissues involved. Cosmetic reconstruction, however, while it should not be delayed unnecessarily, can be deferred for 6 months to a year until the chance of local recurrence begins to diminish and the condition of the local tissues is such that they are more adaptable to successful reconstruction.

Figure 7. When a portion of the mandible is removed primary bony reconstruction has been unsatisfactory in our hands. A Webb bolt will maintain the position of the divided mandible satisfactorily for at least many months if not longer and bony reconstruction can then be undertaken in a sterile field with the removal of the bolt.

The actual technique for monobloc procedures as well as for neck dissections has been outlined in detail elsewhere.[14, 20, 22, 23] Similarly, certain precautions to minimize intraoperative and postoperative complications have been described.[24] With the more widespread use of preoperative radiation and a more frequent surgical acceptance of an effort to salvage radiation failures, skin sloughs may develop which can progress to carotid blow-outs. Incisions which will avoid a trifurcation at the carotid bifurcation are helpful in preventing this complication. It is also useful to transplant viable tissue over the carotid artery. We generally use the levator scapulae muscle, though others have used the trapezius muscle[11] or a dermis graft.[5]

RADIATION MANAGEMENT

It is not within the scope of this article to present detailed descriptions of variations in radiotherapeutic techniques. In the last 20 years, however, just as there have been modifications in surgical techniques, there have been vast changes in radiotherapeutic modalities with resultant improvement in salvage rates.

The biggest and most significant change has been the widespread use of supravoltage irradiation equipment, particularly cobalt-60.[16] Low intensity radium needles are also more widely used now than they were at one time. Other radioactive materials include radon seeds and interstitial iridium-192.[16] The iridium tapes originally utilized were made of a plastic material and not infrequently broke. More recently iridium pins have been used, much on the order of the low intensity radium needles except that they are after-loaded through stainless steel guides. There is some tendency for these interstitial modalities to become loosened and migrate altering the planned dosage schedule. Supplemental external irradiation is then necessary.

ELECTIVE NECK DISSECTIONS

Kremen was one of the earliest to advise a routine neck dissection preferably by the monobloc technique in the treatment of cancer of the tongue.[14] When reviewing our own material at that time we found that clinically negative nodes were microscopically positive in just under 40 per cent of the patients (Table 1).[25] It must be admitted however that the majority of the patients in this series had a monobloc procedure. The elective neck dissection was performed because their primary tumor was of such advanced stage that its treatment required opening the neck to obtain satisfactory exposure of the lesion. A somewhat higher incidence of unsuspected metastatic tumor might well be expected in this group of patients. Jesse recently presented some material on the incidence of development of neck metastases which is at variance with this philosophy (Table 2).[12] Regardless of the stage of the primary lesion, there was no significant change in the incidence of the development of subsequent met-

Table 1. *Incidence of Clinically Occult Metastases in Cancer of the Tongue*

STUDY	YEAR	INCIDENCE
Lyall and Shetlin	1952	60%
Kremen	1967	50%
Southwick et al.	1960	39.9%
Sako et al.	1964	27.6%
Ward et al.	1959	25%

astatic cervical node disease. Almost all of the advanced patients in this series, however, received 5000 rads in 5 weeks to the upper neck as part of the treatment of their original intraoral primary. Jesse postulates that this partial radiation of the neck possibly prevents some clinical disease from developing subsequently.

At variance with this, however, was a recent report by Farr and Arthur suggesting a more definite correlation between the size of the primary tumor and the subsequent development of positive regional lymph nodes (Table 3). Specifically all tongue lesions over 2 cm. in diameter had a yield of greater than 50 per cent positive nodes.

It should be made clear in a discussion of elective neck dissection as to whether or not the procedure is to be routine, selected, or never. Harrold,[20] for instance, does not advise "elective neck dissection" and yet 22 per cent of the patients with clinically negative necks and a primary cancer of the anterior two thirds of the tongue in his series have had a neck dissection performed as part of the adequate treatment of the primary tumor. Similarly 61 per cent of the patients with their cancer at the base of the tongue had a neck dissection as part of the management of their primary tumor problem. Likewise, while Jesse and his colleagues do not advise treatment of clinically negative necks, they do increase the size of their radiation ports and also the dosage to the upper neck in patients who might in other institutions have a monobloc procedure.

I feel that *selective* neck dissections are indicated. The selectivity must take into consideration the site of the primary tumor (which includes the tongue), the size of the primary tumor (which should be at least 2 cm. or greater), the infiltrating rather than the exophytic tumor,

Table 2. *Incidence of Cervical Node Metastases after Partial Glossectomy*

STAGE	NUMBER OF PATIENTS	NUMBER IN WHOM METASTASES DEVELOPED	PER CENT
T_1	169	39	23
T_2	200	52	26
T_3	110	32	29

Table 3. *Incidence of Cervical Node Metastases after Partial Glossectomy*

STAGE	NUMBER OF PATIENTS	NUMBER IN WHOM METASTASES DEVELOPED	PER CENT
I	95	28	29.1
II	77	33	42.9
III	13	10	77.0
Total	185	71	38.4

and finally, the likelihood of successful follow-up examination. This latter point is particularly important in certain underdeveloped areas of the world.[21]

COMBINATION THERAPY

The use of preoperative radiation and its effectiveness in reducing local recurrence in the neck has already been noted. While such local recurrence may be reduced, however, there is as yet insecure evidence that the overall salvage rate will be improved by such a combination of therapy.

Additional combination therapy has been undertaken using chemotherapy in conjunction with either surgery or irradiation. In general, chemotherapy combined with surgery has been used for the palliative management of advanced and otherwise incurable disease. Certain specific protocols, however, have been developed for a combination of radiation and chemotherapy given either simultaneously or metachronously. Chemicals used have been variable, with methrotrexate and 5-fluorouracil most common. At our institution an experimental study using bleomycin is now under way. The chemical agent is being used both as a full course preceding radiation as well as simultaneously with the radiation.[22] The results of such programs to date have not been particularly encouraging as far as any significant improved increments of salvage are concerned.

REHABILITATION

Rehabilitation has two major forms, one functional and the other cosmetic. Some of the considerations of rehabilitation were mentioned earlier in regard to the preoperative planning of a treatment program.

One of the more interesting areas of pretreatment functional rehabilitation is the use of fluoride compounds with special carriers prior to and in the immediate post radiation period. Daly[6] has noted a marked protective influence with the fluoride. Even in a group with good dental hygiene, 65 per cent of those who did not receive fluoride developed post

Table 4. *Overall Survival in Surgical Treatment of Cancer of the Tongue*

AUTHOR	NUMBER OF PATIENTS	5 YEAR SURVIVAL
Kremen	44	70%
Beahrs et al.	80	58.8%

irradiation tooth decay, whereas in the treated group only 22 per cent developed such caries.

Surgical reconstruction either functional or cosmetic is generally undertaken by our plastic surgery colleagues. Certain basic simple primary reconstructive procedures are undertaken by most head and neck surgeons but care should be exercised at the time of primary reconstruction so as not to jeopardize optimal cosmetic reconstruction. Temporary wires, bars, and so forth are often useful in maintaining functional rehabilitation and temporary cosmetic acceptability. In spite of the fact that we find that there is much more common widespread distribution of metastatic disease from the tongue than it was at one time believed,[18] it must be remembered that the patients generally die of failure of control in the head and neck area and not of the disseminated disease. The fact that such dissemination does occur should not deter one from undertaking certainly functional if not cosmetic reconstructive surgery.

RESULTS OF TREATMENT

Kremen's statistics still present the best five year survival free of disease for all cases of tongue cancer including both the mobile and the posterior aspect of the tongue (Table 4).[15] While the series was small (44 cases) 70 per cent survived 5 years free of disease and 44 per cent of those whose nodes were clinically and microscopically positive survived 5 years. Beahrs et al.[2] reported 58 per cent 5 year survival in the surgically treated patients. This was a selected series, however, as the lesions at the base of the tongue were often treated with radiation rather than with surgery.

Two large series of patients treated with radiation have recently been reported.[1, 7] Each had a 5 year survival just under 35 per cent (Table 5). It

Table 5. *Overall Survival in Radiation Treatment of Cancer of the Tongue*

AUTHOR	NUMBER OF PATIENTS	5 YEAR SURVIVAL
Ash	671	34.8
Easson	798	33.5

Table 6. *Cancer of Tongue, Control at Primary Site*[12]

	SURGERY	RADIATION
T_1	86%	86%
T_2	93%	79%
T_3	63%	50%
T_4	50%	34%

must be remembered however, that these figures represent total experience and include a higher percentage of the posterior third (and less favorable) lesions than the surgical series.

As might be expected the salvage rates for the anterior two thirds or mobile portion of the tongue are considerably better than for the posterior third. They are almost identical whether the method of management is primarily surgery or radiation (Table 6). Spiro and Strong[29] recently reported a series of 185 patients who had a partial glossectomy for localized epidermoid cancer of the tongue. The 5 year salvage rate of determinate cases was 62.1 per cent. Control of the primary tumor was accomplished in over 80 per cent of the lesions T_2 or smaller. Neck dissections in this group were reserved for those patients who showed clinical evidence of disease and 77 per cent of the patients with T_3 lesions demonstrated such disease. There was a failure rate of 60 per cent in the patients who required therapeutic neck dissections. Selective neck dissections might have improved this salvage.

The best results with radiotherapy for anterior tongue lesions were achieved through the use of interstitial radiation. Pierquin and the M. D. Anderson Hospital both report high degrees of local control.

SUMMARY

The management of tongue cancer by competent radiotherapy or surgery can produce reasonably satisfactory salvage rates for the mobile tongue when the lesion is T_1 or T_2. The larger lesions of the mobile tongue and posterior third and base of tongue have a much poorer prognosis. This area particularly requires imaginative joint treatment programs in an effort to improve the survival rate. Selective neck dissections are indicated.

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Carcinoma of the Larynx

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In the past 150 years carcinoma of the larynx has had an interesting and controversial history[6, 14] (Table 1). The adoption of the laryngeal mirror by Manuel Garcia, who developed it from previous dental mirrors, made the study of carcinoma of the larynx clinically possible. Garcia was a music teacher who used his mirror to demonstrate the vocal cords. He introduced this mirror into the medical literature, and was well known by physicians of Paris and London during the middle part of the nineteenth century. His observations were noted by the laryngologists of his day, and his mirrors were used to aid the taking of biopsies by indirect laryngoscopic examination. This mirror undoubtedly led to the performance of the first laryngectomy, which was done in 1866 by Watson of Edinburgh. Like most laryngectomies in the nineteenth century, it was not performed for carcinoma but for granulomatous disease.

The great Viennese surgeon, Theodore Billroth, performed the first laryngectomy for carcinoma in 1872. Survivals for carcinomas were rare, since early surgeons did not have the advantages of intravenous fluids, tube feedings, and blood transfusions; and the unsutured pharyngeal stomas were left to close by secondary intention.

Later, Chevalier Jackson refined peroral endoscopy to facilitate the diagnosis of these lesions, and Crile in 1906 suggested the importance of doing a neck dissection in association with carcinoma of the larynx. At this time radium was first used for therapy. Irradiation was later refined in about 1920 when orthovoltage and in 1953 when cobalt and megavoltage radiation were introduced.

In 1851, an American surgeon, Gordon Buck, performed a laryngofissure for a carcinoma of the larynx, this undoubtedly being one of the first "partial laryngectomies." It was not, however, until about 1930 that a Uruguayan surgeon, Justo Alonso, departed from what was then classical

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Table 1. *Laryngectomy: Historical Review*

1833	First laryngofissure for tumor treated with cautery.
1851	First laryngofissure for excision of tumor.
1854	Indirect laryngoscopy discovered by Spanish singing teacher, Manuel Garcia.
1866	First laryngectomy performed by Watson of Edinburgh for syphilis.
1871	Morell MacKenzie, indirect laryngeal biopsy.
1873	First laryngectomy for carcinoma performed by Billroth.
1878	Billroth performs the first conservative hemilaryngectomy.
1880–1900	Operative mortality of total laryngectomy nearly 50 per cent.
1887	Morell MacKenzie and Crown Prince Frederick of Germany. Biopsy by indirect laryngoscopy benign but the patient soon died of his malignancy.
1895	Roentgen discovers x-rays.
1898	The Curies discover radium.
1906	Crile suggests the importance of lymphatic dissection for head and neck tumors.
1907	Radium first used in therapy.
1915	Lynch suspension laryngoscopy.
1915–1920	Development of orthovoltage 200 to 450 kv radiotherapy.
1922	Coutard treats advanced laryngeal malignancy with fractional radiotherapy.
1925–1940	Surgery predominately for early intrinsic cancer with radiotherapy for advanced cases.
1930–1940	Introduction of supervoltage radiotherapy.
1940–1960	Radiotherapy as treatment of choice for early superfocoa; cancer with surgery for the more advanced cases.
1941	First production of cobalt-60.
1951	Standardization of the surgical technique now employed for laryngectomy and neck dissection.
1951	Cobalt-60 first used for teletherapy.
1961	Preoperative irradiation.
1962	Clinical staging system for cancer of the larynx.

**From* Rosenberg, P. J.: Arch. Otolaryngol., 94:313–316, 1971; by permission. Copyright 1971, American Medical Association.

total laryngectomy by performing partial vertical and horizontal laryngectomies. His work was continued by Joseph Ogura and Max Som in the United States. All three have subsequently shown that the cure rate for partial laryngectomies on properly selected cases equals that of the total laryngectomy, with the obvious advantage to the patient of preservation of function. Early detection of cancer and partial laryngeal (conservation) surgery thus play an important part in the present day diagnosis and treatment of laryngeal cancer.

Etiology

Of the 243 patients with laryngeal carcinomas, evaluated, treated, and followed for a minimum of 2 years at the University of Chicago, at least 186 were cigarette smokers with an average duration of 31 years of smoking history. A smaller number of patients were also cigar and pipe smokers and it was not unusual for the latter two groups to inhale the smoke. A smaller percentage of the patients were alcoholics. It is felt that, without question, smoking plays an important role in the development of this disease and greatly increases the chances of recurrence. We

agree with Moore[8] who reports that the incidence of local recurrence and the appearance of second primary lesions are high in individuals who continue smoking.

Diagnosis and Symptoms

Most lesions are identifiable by an indirect laryngoscopic mirror examination and, for that reason, any patient with hoarseness beyond 2 weeks or persistent pain in the throat or dysphagia should have at least this examination. We consider that a preoperative laryngogram (Fig. 1) performed under local anesthesia with Dianasol along with an esophagram and a carefully performed direct laryngoscopy should be included in the preoperative work-up of all these patients for more correct staging of the tumor. In all instances a positive biopsy is required. A tomogram is acceptable if laryngograms are not available. A chest x-ray and bone survey should also be included in the work-up since second primaries, in particular lung cancer, are common. The success of total laryngectomies or conservation surgery (partial laryngectomy) depends on the precise staging of the laryngeal tumor. Recently an operating microscope with a 400 mm. lens has been helpful for diagnostic purposes.

We recently evaluated our experience with laryngeal tumors followed for 2 to 25 years from 1937 to the present day. This covered a period of great change in radiation therapy and surgical techniques at the University of Chicago. For purposes of discussion, lesions will be divided

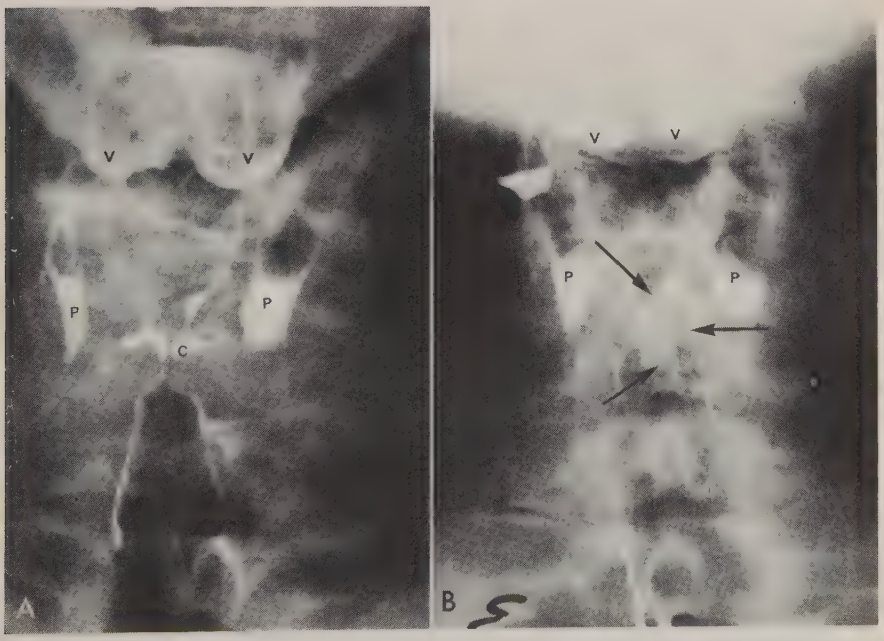

Figure 1. *A*, Frontal laryngogram performed with Dianasol under local anesthesia. The vellacula (*V*), pyriform fossa (*P*), and the vocal cord (*C*) are easily seen. *B*, Frontal laryngogram in a patient with a transglottic lesion of the left side. The vellacula (*V*) and pyriform fossa (*P*) are seen. Arrows outlines the size of the carcinoma of the larynx.

into groups from T_0 to T_4 and subdivided into supraglottic and glottic regions. Subglottic lesions are not included since these were present only in a small number in our series, and large hypopharyngeal lesions were not considered in this study. Staging was defined clinically by mirror examination, direct laryngoscopic examination, and examination of the neck. The Joint Committee on Cancer Staging and End Results Reporting (1962) was used as a guide for defining the stage.* Almost all the lesions were squamous cell carcinomas.

T_0 Lesions

These lesions are defined as carcinoma in situ. Pathologists differ as to the precise definition of carcinoma in situ, but in our institution it is defined as cytologic changes of malignancy without invasion of the basement membrane. Individuals with carcinoma in situ will quite frequently develop an invasive carcinoma, especially if smoking habits are continued. Opinion on the best treatment for these lesions is divided between local excision (vocal cord stripping) and irradiation. Alden Miller[7] describes a large series of treated patients, of whom 16.5 per cent develloped an invasive squamous cell carcinoma. He recommends treatment by stripping of the involved vocal cord in localized lesions. In his experience, if the lesions are more extensive or involve both cords, irradiation is recommended. Irradiation is also recommended for recurrence of carcinoma in situ following vocal cord stripping. Our methods are similar to his, and radiation is given to those lesions which are extensive in nature, bilateral, or recurrent after vocal cord stripping.

T_1 Lesions (Glottic)

A T_1 glottic lesion is defined as a tumor confined to one vocal cord where the mobility of the cord remains normal. T_1 glottic lesions grow slowly, are usually well differentiated, and metastasize late because the vocal cords have a limited lymphatic supply. In many institutions T_1 lesions are quite successfully treated by surgical methods, most frequently by laryngofissure or cordectomy. This lesion also can be handled by radiation therapy alone, with surgery reserved for biopsy proven recurrences. This allows better preservation of laryngeal function and provides a cure rate similar to that of the surgically treated group. We have treated a total of 49 cases (see Table 2) of T_1 lesions in the glottic group. All the laryngofissures were done prior to 1956 and the laryngectomies were done prior to 1947. Recurrence rate with irradiation alone was 24 per cent (9 out of 38 cases). Of the 9 patients with recurrences, 5 patients were later salvaged with partial or total laryngectomy and 2 with laryngofissures. It was previously thought that a total laryngectomy was necessary after a radiation failure, but Norris,[9] Balantyne,[1] and others now consider that a partial laryngectomy for the radiation failure is both possible and curative in properly selected cases. It is important that these patients be followed on a regular basis after irradiation. Twenty-four per cent recurrence rate after irradiation is high, but we are confident that with surgery salvage from the original group a respectable cure rate can be obtained.

*This article was prepared before the new staging system of the American Joint Committee was published (July 1972).

Table 2. *Treatment and Recurrences in Laryngeal Carcinoma without Node Involvement*

TYPE OF LESION	TOTAL	RADIATION THERAPY	SURGERY	COMBINED	RECURRENCES	
					Radiation Therapy	Surgery
T_1 Glottic	49	38	11*		9	3
Supraglottic	11	8	3		2	0
T_2 Glottic	61	40	18	3	12	5**
Supraglottic	10	3	5	2	0	1
T_3	60	20	31	9	11	9**

*Laryngectomies and laryngofissures.
**Includes cases in the combined group.

T_1 Lesions (Supraglottic)

T_1 supraglottic lesions are defined as lesions limited to the laryngeal surface of the epiglottis, or to the aryepiglottic fold or the arytenoid, or to a ventricle cavity or band. T_1 supraglottic lesions are treated similarly to the T_1 glottic lesions in that a curative dose of radiation therapy is given, i.e., 6500 rads in 6.5 weeks (see Table 2). It must be kept in mind that, unlike the T_1 glottic lesions, supraglottic lesions have a tendency to metastasize early, and careful attention must be directed to the evaluation of metastatic neck nodes. Normally, a prophylactic neck dissection is not done for either the T_1 glottic or T_1 supraglottic lesions.

T_2 Lesions (Glottic)

T_2 lesions are defined as tumors involving both cords with normal mobility of cords; or tumor of both cords with limited mobility of both cords. A total of 71 cases were treated in the T_2 category (see Table 2). Of these, 40 were treated with radiation alone with success in 28 cases. Of the 12 radiation failures, 8 were later cured with surgery. Of the cases treated only with surgery, the cure rate was 18 of 23.

Biller[7] and others advocate partial frontolateral laryngectomies for T_2 cord lesions (Fig. 2). He reports a 3 year survival rate of 77 per cent. Surgery offers an excellent prognosis in this category; however, the T_2 glottic lesions are treated at this institution initially with radiotherapy, reserving surgery for the radiation failures.

T_2 Supraglottic Lesions

T_2 supraglottic lesions are defined as tumors of the epiglottis extending to the ventricular bands. This group can be treated in the same way as the T_2 glottic lesions with primary curative radiotherapy of 6500 rads in 6.5 weeks, reserving surgery for the radiation failures. In the pioneer work of Ogura, Som, and Alonso, it was shown that partial horizontal laryngectomies are as successful as total laryngectomy for these lesions and have the obvious advantage of preserving function (Fig. 3). A total of 10 cases in this category have been treated (see Table 2).

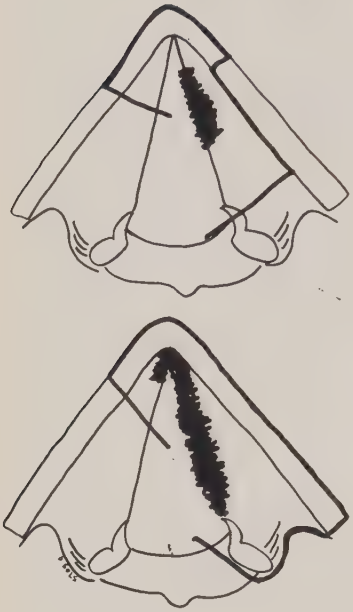

Figure 2. Two versions of a frontolateral hemilaryngectomy, dark lines showing area to be excised. (*From* Norris, C. M.: Cancer of the Larynx: A Monograph for the Physician. Published by The American Cancer Society, 1963, reproduced with permission.)

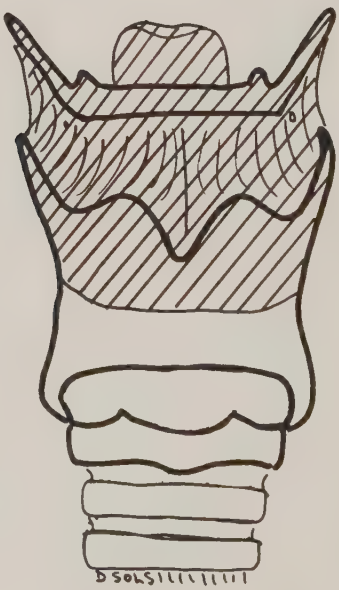

Figure 3. Horizontal hemilaryngectomy for supraglottic cancer. Striped lines show the area to be excised.

Table 3. *Recurrences in Laryngeal Carcinoma with Node Metastases*

	NUMBER OF CASES	RECURRENCES
T_1 with Nodes	11	9
T_2 with Node(s)	4	2
T_3 with Node(s)	12	5
T_4 with Node(s)	21	19

T_3 Lesions (Glottic and Supraglottic)

T_3 lesions are defined as tumors extending from cords either to the subglottic region or to the supraglottic region. The supraglottic area comprises tumors of the epiglottis and/or ventricles or ventricular bands, and extending on to the cords. No difference was found in the 3 year cure rate if preoperative irradiation was given; consequently, T_3 lesions are not given preoperative irradiation. The recurrence rate for T_3 lesions is given in Table 2.

T_4N_0 Lesions

T_4's are lesions or tumors extending beyond the larynx, with direct extension through cartilage, skin, or post cricoid region. In this group, 1 of 4 patients was cured with primary treatment, and an additional patient was cured with a secondary surgical procedure, giving us a salvage rate of 25 per cent. At the present time T_4N_0 lesions are given preoperative irradiation followed by surgery.

Nodal Metastasis

Nodal metastasis requires a neck dissection and the prognosis immediately becomes worse (Table 3).

Staging

It is interesting to note that the pathological staging after surgery correlated with the preoperative staging in our series 64 per cent of the time. In 22 per cent of our cases, the pathological staging revealed a more advanced tumor than was clinically expected and in 13 per cent the staging revealed a smaller tumor than was clinically expected.

Recurrences

For glottic cancer, the number of biopsied recurrences which occurred within 2, 3, and 5 years for each stage is shown in Table 4. It

Table 4. *Late Recurrence of Glottic Cancers*

	BIOPSIED RECURRENCES	WITHIN 2 YR.	WITHIN 3 YR.	WITHIN 5 YR.
T_1N_0	12	5	7	10
T_2N_0	19	10	11	16
T_3N_0	9	8	8	8

seemed significant and unusual that only 5 of 9 of the T_1N_0 cases and 6 of 12 of T_2N_0 cases showed recurrences within 2 years since it was expected that the majority of the recurrences would appear before 2 years. It was not, however, until 5 years after treatment that 80 per cent of the cases showed recurrences. Horiot[5] also reported the late recurrence of glottic cancers, but to a lesser degree, since 66 per cent of his recurrences occurred within 2 years. Supraglottic cancer, on the other hand, has a tendency to recur within 2 years.

Complications

From a total of 93 cases treated by radiation alone, cartilage necrosis developed in only 1 case. This was a patient with systemic lupus erythematosus. Data on voice quality and arytenoid edema were in general lacking. Twenty-six of the surgically treated cases developed pharyngocutaneous fistulas. Stenosis of the esophagus developed in 6 patients and the skin flap sloughed in 2. Many of these had received high doses of preoperative radiation.

Discussion

In evaluating laryngeal lesions a number of pertinent questions have arisen. The first is whether to use irradiation or surgery as a primary mode of therapy. Most certainly in the T_1 lesions and the greater majority of the T_2 lesions irradiation should be offered as a treatment of choice, unless the general health or the cooperativeness of the patient dictates otherwise. The value of preoperative irradiation is not that well known, especially for the T_3 transglottic lesions. In a large series of cases Ogura[10] showed that low dose preoperative irradiation (1000 to 3000 rads) benefits pyriform sinus and laryngeal pharyngeal lesions. Preoperative irradiation is also discussed by Bryce.[3] These reports are in contrast to that of Goldman[4] who described an increase in the cure rate when a dose of 5500 rads of cobalt therapy was given preoperatively. There is evidence that the large T_4 lesions of the larynx which extend to the hypopharynx or through the cartilage to the pre-epiglottic space are helped with preoperative irradiation. Specifically, recurrence rates are less and the survival is greater. Postoperative irradiation is not given on any regular basis except for those tumors that cannot be adequately removed with good margins, and in patients where surgery was done first because of their unreliability.

A good percentage of the recurrences appear after 2 or 3 years, especially for the glottic group. Furthermore, patients who received curative doses of irradiation frequently had recurrences after 3 years.

It has been traditional in laryngeal surgery that a prophylactic neck dissection be done for large laryngeal tumors. Recently, however, there have been evidence and opinions that a prophylactic neck dissection is not always indicated, especially for smaller transglottic lesions.[11-13] There is obviously a need for re-evaluation in this area, but the indications for so-called prophylactic neck dissections appear to be changing.

We continue to do prophylactic neck dissections for large transglottic lesions and T_4 lesions extending beyond the confines of the larynx. Second neck dissections are frequently performed if multicentric metastases are present in the original neck. The lateral "T" incision has been useful

in protection of the carotid artery. Dermal grafts and levator scapulae muscle transpositions are also useful.

SUMMARY

T_0 glottic lesions are best handled by local stripping unless both cords are involved and extensive disease is present. The latter two conditions are treated with 6500 rads in 6½ weeks. T_1 glottic and supraglottic lesions should be given a curative dose of radiation (6500 rads in 6½ weeks). Recurrences may be treated with partial or total laryngectomy. Neck dissections are not usually done in these groups.

T_2 glottic lesions are best handled initially by irradiation, followed by surgery for the radiation failures. We are not in disagreement with partial laryngectomy for preservation of function in this group.

T_2 supraglottic lesions are handled initially by irradiation (6500 rads) unless the lesion is large, in which case we would recommend that surgery be done first.

T_3 transglottic lesions are usually handled by surgery alone without preoperative irradiation. In many instances, a neck dissection is performed.

For large T_4 lesions preoperative irradiation (4000 rads) followed by surgery in 4 weeks is given for pyriform fossa lesions, hypopharyngeal and post cricoid lesions.

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Carcinoma of the Thyroid

Modalities of Therapy

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Thyroid carcinoma is treated by a range of operative and nonoperative measures which vary according to the histologic type, extent of spread, and personal bias of the physician. Operative measures used are excisional biopsy, thyroid lobectomy, or total thyroidectomy, which may or may not be combined with neck dissection. Nonoperative measures include thyroid hormone suppression, local external irradiation, and systemic iodine-131.

The biologic characteristics of the histologic types of thyroid cancers have been well described.[10, 15] There can be no doubt that papillary and follicular carcinoma of the thyroid pursue a much more benign course than that of anaplastic carcinoma.[9] These lesions, therefore, will be discussed separately, with regard to therapy.

Thyroid carcinoma may present clinically in any of three stages of growth which require different measures for proper management. The first stage consists of those tumors which are confined to the gland itself. The second stage has extracapsular extension but is still confined to the local tissues and lymph nodes in the neck. The patient in stage three has developed disseminated metastases, usually in bone or lungs. Through clinical assessment of the extent of spread of thyroid carcinoma, guidelines of therapy including nonoperative and operative means can be established. Each stage, therefore, will be discussed separately with reference to the particular histologic types.

BIOLOGIC INFLUENCES

The development of thyroid carcinoma in young females is well known, being approximately 20 to 40 per cent of all cases.[15, 18, 19] Since 1950, many reports of thyroid carcinomas induced by externally applied irradiation have been published. This irradiation was usually given

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for thymic or tonsillar disease to young children. However, recent cases of thyroid carcinoma induced from irradiation for acne have been reported.[19] Yet to be determined, however, is the carcinogenic effect of iodine-131 given for the treatment of hyperthyroidism. To this time, no study has come forth linking this disease with iodine-131 therapy.

In patients over 40 years of age, thyroid carcinoma has been reported to be a much more malignant disease than in younger patients.[5] This finding may simply represent a slow-growing untreated or inadequately treated carcinoma; this possibility has not been thoroughly evaluated.

In a series published by this author, a biologic predisposition was found.[18, 19] A predilection was found for the development of thyroid cancer in patients with blood type A (60 per cent), and a concomitant decrease in those patients having blood type O (18 per cent). The patient groups included those with prior treatment by irradiation and also those with extracapsular extension of their disease.

Therefore, the young female who has external irradiation to the neck has a greater chance of developing this disease especially if she is found to have blood type A.

INTRACAPSULAR CARCINOMA

Following routine clinical evaluation for the suspicious nodule, the usual operation has been excisional biopsy or simple thyroid lobectomy. Pathologic evaluation, either by frozen or permanent section technique, will reveal the carcinoma when present. Complete removal of the entire thyroid gland should then be undertaken.

Using the whole organ section technique, Russell discovered that tumors of the thyroid may extend into the isthmus of the thyroid, the opposite lobe, or even to the pericapsular lymph nodes along the opposite lobe in 87.5 per cent of the glands examined.[12] Although the recurrence rate is not nearly this high following simple lobectomy, subsequent tumor has developed in up to 50 per cent of patients treated by lobectomy alone.[2, 19] Additional advantages ensuing from total thyroidectomy are several. Recurrent disease in the gland is obviated. Early detection of recurrent disease in the neck by use of the iodine-131 scan technique is facilitated. Treatment of disseminated disease with radioiodine is much easier. Transformation from the well differentiated forms of thyroid cancer to an anaplastic form has been reported.[3] This situation is not possible when the total gland is removed.

Occult lymph node involvement can occur in a significant number of patients. If no specific therapy is instituted, over 50 per cent of the patients will develop recurrent disease.[18] During total thyroidectomy an opportunity is afforded to sample the nodes along the carotid sheath. Nodal metastases may be appropriately treated when discovered. Simple lobectomy does not present this opportunity.

With appropriate care, the incidence of permanent vocal cord paralysis and hypocalcemia should be very low. As hormonal suppression is universally used postoperatively, thyroid replacement does not present a significant problem.

The advantage, therefore, of total thyroidectomy for intracapsular carcinoma of the thyroid is total eradication of this frequently multicentric disease. In addition, opportunity to discover occult nodal involvement is afforded as well as the ease of further diagnosis and treatment with iodine-131. These features of treatment together with the simplicity of the operation and the ease of hormonal replacement persuade us that near-total thyroidectomy is the treatment of choice for thyroid carcinoma confined to the thyroid gland itself.

EXTRACAPSULAR CARCINOMA

Carcinoma of the thyroid gland extending to local structures and to lymph nodes in the neck is much more frequent than usually expected, with clinical nodal involvement discovered in over one third of patients at their first examination.[11] The "lateral aberrant thyroid" of the 1950's was a lymph node involvement from an undiscovered thyroid carcinoma. Occult cervical nodal involvement may be present in 20 to 30 per cent of the patients who are clinically negative.[7, 17] This disease complex carries a significant mortality rate with a 5 year survival of 83 per cent.[18] However, the 10 year survival figure is only 61 per cent, demonstrating the malignant nature of metastic thyroid carcinoma.[18]

Because of the frequency of lymph node involvement and the significant mortality from thyroid cancer, most surgeons favor a standard neck dissection in addition to thyroidectomy as the treatment of choice for patients with extracapsular extension of the tumor. The routine application of the radical neck dissection to all patients with papillary carcinoma clinically confined to the thyroid gland has been found to be unnecessary.[8]

The application of the standard radical neck dissection as the treatment of all extracapsular lesions is based on the surgeon's desire to perform an "en bloc cancer operation." Thyroidectomy and neck dissection do not fulfill the usual criteria of a "cancer operation." This procedure is simple organ removal with excision of the regional nodal basin. The thyroid gland, larynx, and esophagus, together with the accompanying vessels and nerves, lie in the visceral compartment of the neck, while the nodes removed by the standard radical neck dissection are contained in the compartment enclosed by the deep and superficial cervical fascia.[4]

In addition to not fulfilling the criterion of a "cancer operation," the standard neck dissection is a disfiguring and partially disabling procedure. The modified neck dissection which spares the sternocleidomastoid muscle presents a reasonable alternative to the standard radical neck dissection. This operation produces a cosmetically superior result in these patients who are frequently young females. The cure rate by modified dissection was 80 per cent, while the standard radical neck dissection cured 70 per cent.[18] This attests to the safety of the operation. Crile has attested to the worth of this procedure: "Modified radical neck dissection appears to be a highly effective way of removing nodes in the area that is actually covered by the dissection."[3] The method for this procedure has been adequately and thoroughly described elsewhere.[1]

When the cervical lymph nodes are involved with carcinoma of the thyroid, discovered either clinically or by sampling the nodes of the carotid sheath during total thyroidectomy, excision of the nodes together with the entire gland should be accomplished at the earliest opportunity. Because of the high occurrence and recurrence of the tumor in these nodes, lymphadenectomy is necessary to prevent the recurrence of tumor as well as to prevent the invasion of the vital structures in the neck. The modified neck dissection, sparing the sternocleidomastoid muscle, can be undertaken providing not only a reasonable expectation for cure, but also a cosmetically superior result.

TREATMENT OF THYROID CARCINOMA ACCORDING TO HISTOLOGIC TYPE

The foregoing discussion is applicable to all tumors of the gland localized to the neck. A very high 5 year survival rate with this method can be expected with papillary carcinoma (96 per cent), a similar rate with follicular carcinoma (98 per cent), and a moderately successful rate with medullary carcinoma. Medullary carcinoma is an unusual disease in the thyroid gland but seems to be of moderate virulence.

Anaplastic carcinoma of the thyroid is a difficult tumor to eradicate; a 5 year survival causes the physician to review the pathologic material. Lev et al. presented a series of 83 patients with anaplastic carcinoma of the thyroid with only 2 patients surviving 5 years.[9] An attempt should be made operatively to clear the tumor from around the area of the trachea, as local invasion and tracheal obstruction frequently result in death.[18]

Therefore, the treatment as outlined for papillary, follicular, and medullary carcinoma should be undertaken with total thyroidectomy for disease in the thyroid gland with accompanying modified neck dissection when necessary. However, the treatment of anaplastic carcinoma should be moderately aggressive with clearing of the tracheal area when possible. No other satisfactory treatment has been developed.

TREATMENT OF METASTATIC THYROID CARCINOMA

In the treatment of metastatic disease from the thyroid carcinoma, radiotherapy, iodine-131, and thyroid hormone have been used. Radiotherapy has been successfully used for local bone disease. Anaplastic small cell carcinoma may be radiosensitive and deserves treatment. Although radiation therapy has been advocated for local disease for the well differentiated histologic types,[14] papillary carcinoma has de-differentiated into anaplastic carcinoma following this type of treatment.[16] Also, the recurrence of this disease has been found to be nearly 100 per cent when radiation therapy is used as the main modality of therapy.[18]

In treatment of pulmonary metastases, radioactive iodine therapy

has been highly effective in some cases.[18, 19] Complete resolution on chest roentgenogram for many years has been noted. Treatment is most effective if used on well-differentiated follicular carcinoma which has been found to take up iodine-131 during a radioactive iodine scintiscan. Uptake of the tumor can be stimulated by total thyroidectomy and exogenous thyroid-stimulating hormone.

Long advocated by Crile, but still controversial, is the use of thyroid hormone suppression as the sole or main modality of therapy. The basis of this therapy is suppression of thyroid stimulating hormone, thereby causing regression of papillary and follicular carcinoma of the thyroid.[3] The mechanism of this action is due to the lack of autonomy in the well differentiated types of thyroid carcinoma. Because of this theory of therapy, some physicians have advocated this treatment in lieu of more appropriate and well supported operative management. The efficacy of thyroid hormone in addition to appropriate operative procedures is of unquestionable value. However, the use of this treatment as a panacea for all types of thyroid cancer must be condemned. The recurrence rate has been reported to be much higher than that reported by Crile.[18] To withhold appropriate management on the basis of unsupported theory is therapeutic nihilism.

For the treatment of metastatic thyroid carcinoma, iodine-131 can be given for evidence of pulmonary metastases. External irradiation can be used for palliation of local bone metastases. Effective use can be made of thyroid hormone as an adjuvant in the treatment of well differentiated tumors. The use of any of these modalities in and by themselves, however, is to lose a possible chance for cure in the face of more effective methods.

SUMMARY

An outline of therapy for carcinoma of the thyroid has been presented. Possible biologic influences have been discussed. By separating this disease histologically and by extent of spread on clinical grounds, a rational approach can be devised.

Papillary and follicular cancer should be treated similarly in similar stages. For local disease, total thyroidectomy with a sampling of the nodes along the carotid sheath will produce a satisfactory rate of cure. In the presence of nodal involvement whether clinically obvious or occult, the modified neck dissection will suffice to remove the nodal basin without the cosmetic deformity commonly associated with the standard radical neck dissection. These operations should be supplemented with thyroid hormone as replacement as well as suppression.

Distant spread of disease usually is best treated with radioiodine, although external irradiation may be used for bony metastases. The poorly differentiated forms of thyroid cancer are unsuccessfully treated by any modality. However, effort should be made to clear the airway by local resection or tracheostomy to prevent suffocation. Medullary carcinoma is probably best treated in the fashion similar to papillary carcinoma.

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Chemodectomas of the Head and Neck

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Chemodectoma, a tumor of the chemoreceptor system, occurs more frequently about the head and neck than in other regions. The carotid body, the best known of the chemoreceptors, was first described by Von Haller[7] in 1743 as ganglion minutum. Milligan[18] introduced the term chemodectoma in 1950 as a neoplasm arising from the chemoreceptor system. These lesions are notoriously slow growing and their locations result in both diagnostic and treatment difficulties.

The chemoreceptor organs, which are sensitive to chemical and temperature changes of the circulating blood, are a group of histologically similar structures located in various parts of the body: temporal bone (superior ganglion, jugular ganglion, auricular branch of the vagus nerve and tympanic branch of glossopharyngeal nerve); neck (vagal body, glomus intravagal, paratracheal nerve, superior and inferior laryngeal nerves); orbit (ciliary ganglion); and mandible. Other chemoreceptor tissues have been identified in the mediastinum, abdomen, extremities, retroperitoneal region, and lungs. Chemoreceptors arise partly from neural ectoderm and are associated with cranial and autonomic nerves. Studies have suggested that chemoreceptors are histologically similar to taste buds.

Chemodectoma have been reported associated with thyroid carcinoma and histologically similar tumors of the pancreas and organ of Zuckerhandl. Multiple lesions and familial tendencies have also been observed. Middle-aged women make up the majority of patients reported. The majority of tumors can be excised surgically. Radiotherapy may be of palliative benefit in nonresectable cases. About 5 per cent of chemodectomas show regional lymph node and distant metastases.

Carotid body tumor, the most common chemodectoma, manifests itself as a mass which is movable laterally but not vertically. Located at the

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bifurcation of the common carotid artery, pressure of the tumor on surrounding structures may cause hoarseness, cough, and dysphagia. A thrill or bruit may be associated with a pulsating mass.

Carotid arteriography reveals information as to the size, vascularity, and extent of the tumor. A capillary blush is highly suggestive of chemodectoma. Both cervical carotid arteries should be studied for effective evaluation of the collateral circulation of the opposite side.

Chemodectomas of the glomus jugulare complex (second largest group) are located in the jugular and middle ear areas. Symptoms of intratympanic glomus tumor usually present as decreased hearing acuity, tinnitus, vertigo, pain, bloody discharge from ear canal, secondary infection after rupture of the tympanic membrane, and isolated facial nerve paralysis. Otological and neurological symptoms occur when the tumor extends into petrotympanum and jugular area.

Glomus jugulare tumors usually produce symptoms of paresis of the ninth, tenth, and eleventh cranial nerves, and middle ear symptoms with extension. Lesions in the roof of the jugular fossa may also involve the twelfth cranial nerve if the mesial hypoglossal canal is involved.

Routine skull roentgenograms, tomography, arteriography of the carotid and vertebral arteries and retrograde jugular phlebography may outline the extent of tumor, bony destruction and tumor vascularity.

The ganglion nodosum and the remainder of the cervical portion of the vagus nerve (glomus intravagal) give rise to vagal chemodectomas. A painless mass behind the angle of the mandible bulging into the pharyngeal wall may produce dysphagia. A differential diagnosis of carotid vessel aneurysm has to be considered. Carotid nerve involvement and the occurrence of other chemodectomas along with malignant changes have been documented.

Arteriographic studies showing a vascularized mass above the carotid bifurcation but not close to the temporal bone strongly suggest the presence of a vagal tumor.

Watza and Kleinnassen[15] have discussed glomus bodies of the superior and inferior laryngeal nerves, respectively. It is suggested that the tracheal glomus bodies may be considered as displaced carotid bodies, but the segmental arrangement of the chemoreceptors would suggest that they are embryologically true glomus bodies corresponding to the brachial arches. These chemodectomas are subglottic masses involving the thyroid, larynx, vocal chords, and hypopharynx. Radiographic studies with plane tomography, and scans of the larynx, esophagus, trachea, and related structures may be of value in suggesting the diagnosis of chemodectomas of the trachea and cervical areas.

Surgical removal is treatment of choice in selective cases. Early control of major vessels, recognition and utilization of a relatively avascular plane if present, and recognition of blood supply to the tumor and early ligation are basic surgical principles in removal of the tumor. Catheter shunts with heparinization are necessary adjuncts for excision of chemodectomas associated with the carotid arteries. Hypothermia, anesthesia, adequate hydration, and corrected blood volume during surgery after preoperative collective circulatory evaluation are essential in decreasing postoperative central nervous system deficit.

SUMMARY

Chemodectomas of head and neck have been discussed with salient points being emphasized. Tumors of the chemoreceptor system should be a part of the differential diagnosis when a mass presents in the described areas within this article.

Arteriograms and venograms are useful in diagnosing, locating, and determining the extent of the lesion. Surgical excision of the tumor is the treatment of choice in selective patients. The process of removal is tedious and difficult. Radiotherapy may be of palliative benefit in nonresectable cases.

Hypotensive and hypothermia anesthesia are important adjuncts to surgery.

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Radical Neck Dissection in Carcinoma of the Head and Neck

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Neck dissection for control of cancer of the cervical lymphatic chain was first reported in the late nineteenth century. In 1875 Von Langenbeck performed a combined neck dissection and wide field laryngectomy. A T incision was employed for removal of the larynx, epiglottis, hyoid, part of the tongue and esophagus, both submandibular glands, and lymphatic tissue from the submandibular triangle and lateral neck. Not until 1906, however, when George Crile, Sr., put forth the concept of "en bloc" radical neck dissection in continuity with a removal of the primary lesion was the procedure well described. The rationale followed that of the Halstead radical mastectomy for cancer of the breast. Until the World War II period, general acceptance and use of the operation was delayed. Endotracheal anesthesia and whole blood replacement, coupled with relatively poor cure rates from radiotherapy, provided the impetus for the widespread application of neck dissection to patients with cancer of the head and neck.

Patients with or thought to have metastatic disease in the cervical lymph nodes, patients in whom the lateral neck must be entered to remove the primary lesion, or patients with a primary malignancy of the neck are candidates for the surgery. Distant metastases, incurability of the primary tumor, or nonresectability of the metastatic disease are the main contraindications to neck dissection. The surgeon's aim is complete removal of all cervical lymph nodes and lymph-bearing tissue. To accomplish this, resection of the sternocleidomastoid, omohyoid, and posterior digastric muscles, submandibular gland, tail of the parotid, internal jugular vein, and spinal accessory nerve is necessary. The vagus,

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phrenic, and hypoglossal nerves usually are saved, along with the carotid artery.

Neck dissection may be classified as either therapeutic or elective. When a patient presents with a known primary lesion and enlarged lymph nodes are palpable and thought to contain metastatic cancer, or when the planes of the neck must be violated to remove the primary tumor, the dissection by definition is therapeutic. When no nodes are palpable, but the nature or location of the primary lesion is such that "occult" metastases are likely to be present, if a neck dissection is performed it is called elective or prophylactic.

Considerable controversy surrounds the use of elective or prophylactic neck dissection. Depending on the location of the primary lesion, the incidence of occult metastases from a cancer of the head and neck region is between 0 and 80 per cent. Proponents of elective neck dissection reason that the occult metastases will flourish and become clinically evident, necessitating neck dissection at a later date, but associated with a diminished cure rate. No convincing statistically valid evidence is available to support this thesis.[14] The relationship between the host's immunologic mechanisms and the eventual fate of the occult metastases is far from understood. Those who advocate elective neck dissection usually recommend its use when the incidence of occult metastases to the neck nodes is 30 per cent or higher. Presently we support this approach.

Another indication for a neck dissection is metastases to a cervical node from a primary lesion which cannot be located. Any mass in the neck in an adult, especially if located in the lateral neck and if the patient is a man over the age of 50, most likely is metastatic carcinoma. A careful search for the source of the metastases will identify the primary lesion in the head and neck region 90 per cent of the time. In about ten per cent of patients presenting with a neck mass, no primary cancer will be found at the initial work-up. The primary lesion will eventually surface in 30 per cent of the patients. Among these patients, the primary lesion will be located above the clavicle in four of five. Thus, in such patients, a thorough evaluation of the head and neck is mandatory. The scalp and skin of the face, mouth, oropharynx, hypopharynx, and larynx must be competently evaluated. X-rays of the chest and paranasal sinuses are indicated. Systematic biopsies of Waldeyer's fossa give a 5 per cent yield when the preceding evaluation and studies are negative.

When the primary lesion cannot be located, a needle or open biopsy of the mass is necessary. Properly done, needle biopsies do not give false-positive results for carcinoma and yield clinically valuable information 80 per cent of the time. When an open biopsy is performed, frozen sections must be obtained. The patient and surgeon must be ready to proceed immediately with a radical neck dissection should the mass prove to be squamous cell carcinoma. Since 15 per cent of occult metastases are from the thyroid gland, the surgeon must be prepared to deal definitively with that organ. The 5 year survival rate in all patients with occult metastases to the lateral neck is 25 per cent. Of those masses which eventually prove to be distant metastases from below the clavicle, over 95 per cent are located in the supraclavicular area.

Bilateral neck dissection is indicated in patients with a potentially curable primary lesion and clinically positive but resectable nodes in each neck. Because of an increase in morbidity (up to 60 per cent) and mortality (up to 14 per cent) when a bilateral dissection is performed simultaneously with removal of a primary lesion in the oral or pharyngeal cavity, a 3 week delay between the initial primary neck dissection and the second neck dissection is wise.[9] Only if clamping of the second internal jugular vein at the time of surgery causes significant alteration in the patient's vital signs is the second internal jugular vein preserved. Elective neck dissections for the second side of the neck are not recommended. Only if and when suspicious nodes arise in the second side do we proceed with dissection.

If the cervical nodes are firmly adherent to the internal or common carotid artery, but the primary lesion is resectable, the common carotid artery on the involved side is ligated approximately 1 week prior to definitive surgery. Although this procedure carries with it a mortality of approximately 10 per cent, it does allow for resection of otherwise death-causing disease. Ligation of a common or internal carotid artery in a nonelective situation greatly increases morbidity and mortality.

During the past few years considerable interest and many positive statements concerning the benefits of planned preoperative radiation therapy have been voiced. Although most advocate low dosages, some are convinced that in patients with extensive lesions only cancericidal therapy will improve the cure rates.[3] A most interesting finding associated with one of these studies is that the cancers do not shrink concentrically before the cobalt beam. Usually when carcinoma persists it does so as isolated islands of viable cells throughout the entire area previously occupied by the cancer. Thus, the surgical margins can be no less than those the surgeon would have maintained had the radiation therapy not been given. This makes one question the often heard opinion that an originally nonresectable lesion can be converted with radiation therapy into one that is resectable.

Our experience with tumoricidal preoperative radiation in large lesions has been discouraging. Delay in healing, a discouraging increase in the occurrence of large fistulas that are difficult to close, and an increase in the number of carotid blow-outs occurred—in spite of very careful, gentle surgical manipulation and frequent use of regional flaps—and our cure rates were not greatly improved.

PREOPERATIVE EVALUATION AND PREPARATION

Accurate determination of the extent of the primary lesion and the status of the lymph nodes in each neck is imperative. In addition to a complete examination of the head and neck, a thorough general history and physical examination must be performed. Recent loss of weight, the presence of diabetes mellitus, poor mental status or attitude, and a physiological state significantly greater than the chronological age are important factors in the evaluation. A chest x-ray, electrocardiogram, complete blood count, urinalysis, blood sugar, blood urea nitrogen, and

liver profile should be ordered and reviewed. All patients must stop smoking on admission to the hospital, and any reversible pulmonary disease should be treated. In composite resections carious teeth and gingivitis are possible sources of postoperative infection and must be eliminated prior to surgery. A daily shower and shampoo of the hair with germicidal soap are carried out in the immediate preoperative period.

Although most authors advocate prophylactic antibiotic coverage following surgery, many studies in the literature for general surgical and infectious disease argue effectively against their routine use.[7] Administration of 600,000 units of procaine penicillin every 12 hours starting 24 hours preoperatively, 3,000,000 units of aqueous penicillin intravenously during surgery, and 600,000 units of procaine penicillin every 12 hours for 24 hours postoperatively is our recommendation for patients undergoing composite resection. Such coverage eliminates the threat of an infection by one of the many penicillin-sensitive bacteria found in the pharynx. No antibiotics are given to the patient undergoing solely a radical neck dissection. Specific antibiotic therapy based on culture and sensitivity taken either during surgery or of an infected wound postoperatively seems more logical. The most hazardous infections following head and neck surgery are resistant staphylococcal or gram negative infections; the antibiotics routinely used as prophylactic agents are not effective against these organisms and often predispose to their emergence in the surgical wound.

SURGICAL TECHNIQUE

Radical neck dissection with and without resection of a primary tumor are established procedures[3] and are well described in the surgical literature. While no attempt will be made to detail the specific technique, some aspects of the procedure are especially important and are worth mentioning.

Although we do not routinely use hypotensive anesthesia, we do borrow certain aspects of the technique. The patient's head is tilted up 25 degrees. Halothane is the anesthetic of choice, and positive pressure breathing is continued throughout the procedure. The degree of hypotension achieved with this combination varies from patient to patient, but the overall effect has been a moderate reduction both in the total loss of blood and in the time spent controlling bleeding. Adherence to aseptic technique is mandatory, even if a composite resection is performed. The fewer bacteria contaminating the wound, the better. Proper positioning and draping of the patient is essential. One must be able to move the patient's head and neck from one position to another. Placement of a folded sheet under the middle of the back, use of a head drape, and suturing of the drape into place make this movement possible.

Numerous skin incisions for neck dissections with or without composite resections have been described, advocated, condemned, and rediscovered. The use of increasing amounts of preoperative radiation therapy has heightened the surgeon's concern over the construction and viability

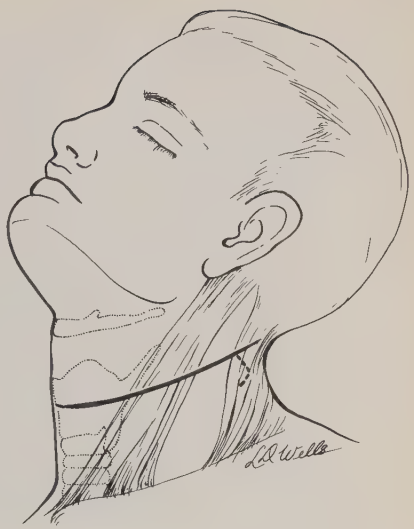

Figure 1. *Incision for neck dissection.* The dotted line shows possible inferior extension if exposure in the posterior triangle is not adequate. Note that the incision does not extend up to the mastoid process but continues posteriorly below it and on to the trapezius muscle. Extension medially is across midline to the anterior border of the opposite sternocleidomastoid muscle.

of the flap. The important considerations to be weighed when deciding on a specific flap are:[6]

1. Unencumbered access to the limits of the surgical dissection.
2. Preservation of the best possible blood supply to the flap.
3. Acceptable relationship of the carotid artery and area of possible fistula formation to the line of incision; e.g., if part of the flap undergoes necrosis, the carotid artery should be minimally exposed, or should a pharyngocutaneous fistula develop, the drainage of this fistula should not further compromise the covering of the carotid artery.
4. Compatibility of the incision with future reconstructive procedures and the flaps.
5. Minimal cosmetic deformity.

The skin incision shown in Figure 1 meets all of these requirements for composite and/or straight radical neck dissection.[2] The initial reaction of most surgeons to the flap depicted in Figure 1 is that the exposure obtained will be inadequate. Fortunately, the skin of the neck is quite lax; thus, the exposure is satisfactory. When reconstruction of the esophagus with a medially based pectoral flap is anticipated, a pair of parallel incisions, one 2 cm. below the mandible and the other 1 cm. above the clavicle work well. Occasionally we cannot determine clinically whether a tumor mass is resectable. Figure 2 shows an approach that has proved worthwhile in such circumstances.

The platysma muscle has few lymphatics in direct continuity with it and is raised with the skin flap. If there is any possibility that the platysma or the skin is invaded by carcinoma, the skin of the questionable area should be resected and a medially based pectoral flap used to close the resulting defect. Sharp dissection against firm vertical countertraction on the flaps facilitates the raising of the skin and platysma. If one is in the correct avascular plane, a thin layer of fascia remains on the ster-

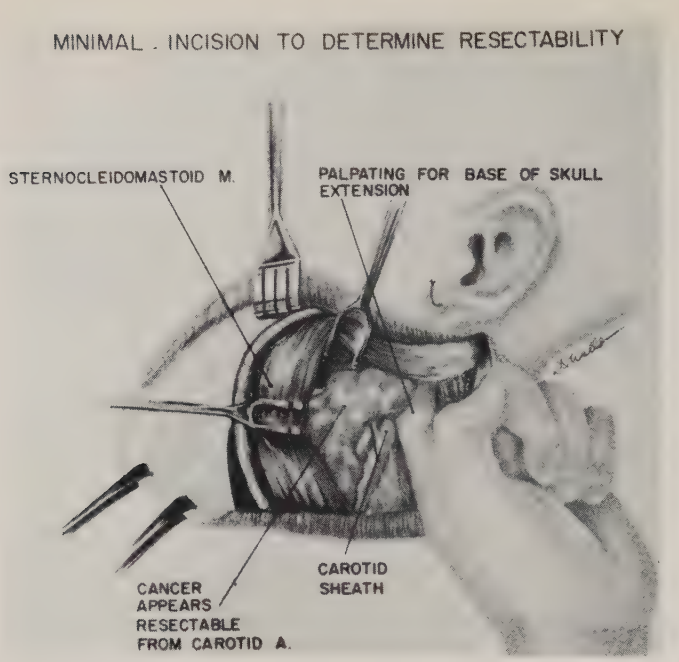
MINIMAL INCISION TO DETERMINE RESECTABILITY
STERNOCLEIDOMASTOID M.
PALPATING FOR BASE OF SKULL EXTENSION
CAROTID SHEATH
CANCER APPEARS RESECTABLE FROM CAROTID A.

Figure 2. When resectability of a large tumor mass is questionable, a relatively small incision allows exploration to determine possible involvement of the carotid artery or extension to the base of the skull.

nocleidomastoid muscle and bleeding is minimal. Frequently one reads that when a surgical field has received previous radiation therapy, special care and diligent meticulous dissection of tissue are mandatory. Certainly we share the apprehension and concern for primary healing in such cases, but we find it difficult to accept rougher handling of tissue or less concern for the quality of the surgical technique in the "routine operation."

At times dissection of the internal jugular vein may prove trying. The middle thyroid vein is the only tributary of the internal jugular in the lower neck. Although absent about 50 per cent of the time, when present it enters on the medial aspect of the internal jugular vein and care must be taken not to tear it. Isolation of the jugular vein is greatly facilitated if manipulation is carried out in a plane directly beneath the loose adventitia surrounding the vessel; dissection should be against the firm vein wall. In the upper neck knowledge that the internal jugular vein lies medial to the posterior belly of the digastric is helpful. Thus, one may use the posterior digastric muscle as a landmark for identification of the internal jugular vein. To adequately clear this area, the posterior belly of the digastric muscle must be included in the specimen.

Blunt dissection against firm upward traction is the key to efficient and rapid clearing of the posterior triangle. A thin layer of fascia must be maintained over the muscles of the floor of the dissection. Branches of the cervical plexus—C_4, C_3, C_2—entering into the specimen are encoun-

tered as one proceeds along this floor. As these are identified and transected, and if one is in the correct plane, the specimen almost floats off the muscular floor.

Should the thoracic duct be entered during manipulation of the lower end of the internal jugular vein on the left, immediate identification of the tear using the operating microscope and closure with nonabsorbable vascular sutures prevent formation of a chylous fistula—a very bothersome problem. Before closure, the wound is irrigated with copious amounts of balanced salt solution. This maneuver improves hemostasis and removes debris that might be a nidus for infection.

Drainage of the neck wound and adherence of the skin flaps are best obtained with two number 18 catheters inserted into the supraclavicular space. Care must be taken so that neither overrides or traumatizes the carotid artery. Another catheter is placed posteriorly along the border of the trapezius muscle. All three are attached to a constant suction apparatus.

POSTOPERATIVE CARE

The patient undergoing a neck dissection alone does not need a tracheostomy. For most composite resections, however, a tracheostomy is mandatory. Careful attention to humidification and aseptic elimination of secretions from the tracheobronchial tree are essential in those patients during the immediate postoperative period. For the first 48 hours the head of the bed is elevated 45 degrees. Within 12 hours of surgery the patient should be sitting at the bedside, and within 24 hours be up and walking. Pain is much less severe following major head and neck surgery than following major abdominal surgery; thus, only small amounts of narcotics are needed postoperatively. The dressing is changed daily until the suction catheters are removed on the third postoperative day. Gloves, gowns, and masks are worn when the dressing is changed. If fluid or blood accumulates beneath the flap, it most often occurs in the supraclavicular area. Prompt aspiration or drainage of any build-up is essential. After the catheters are out, no dressing is necessary. In the patient who has undergone composite resection, the tracheotomy tube is removed when the patient tolerates without respiratory embarrassment progressive decrease in the size of the tracheotomy tube to a number three and is swallowing without significant aspiration.

Physiotherapy and shoulder exercises to compensate for sacrifice of the eleventh cranial nerve are begun on the tenth day following surgery. The patient who has had an uncomplicated postoperative course is discharged from the hospital about the tenth day.

COMPLICATIONS

In head and neck surgery the only sure way to avoid complications is to do no surgery. Numerous kinds of complications occur and may be secondary to poor surgical judgment, faulty surgical technique, the neces-

sary sacrifice of vital structures, virulence of the tumor, poor general condition of the patient, previous radiation therapy, inadequate postoperative care, or inability of the patient to adjust physiologically to his altered physical condition. The complications fall into four categories according to time of occurrence: those occurring during surgery, immediate postoperative, delayed postoperative, and late postoperative complications.

Complications Occurring During Surgery

The most significant complications at surgery are vascular injuries. In order of frequency of injury, the structures involved are the internal jugular vein, the thoracic duct, the subclavian vein, and the carotid artery. If the injury is to the internal jugular vein in the lower neck, one may have to split the clavicle for adequate access. An injury to the upper portion of the internal jugular vein that cannot otherwise be handled can be controlled well with iodoform packing which remains in place for 7 days. Injuries to the other vessels are best handled with routine vascular techniques, although access to the subclavian vein is poor until the clavicle is sectioned and retracted. As stated earlier, a tear in the thoracic duct is best repaired under magnification.

Occasionally air emboli have been reported after injury to the subclavian vein or the lower portion of the internal jugular vein. Prompt pressure over the vein until the injury is repaired usually prevents this complication. An inadvertent injury to any of the nerves in the area of the neck dissection is best repaired primarily with the use of the operating microscope. Postoperative pneumothorax and pneumomediastinum have been reported, but the incidence is very low.

Immediate Postoperative Complications

Bleeding into the wound with hematoma formation is an uncommon but frustrating problem. When it occurs, the patient is returned to the operating room, the incision opened, the clots removed, the wound irrigated, and bleeding areas ligated. Usually a definite source of the bleeding is *not* found. Since such wounds are prone to infection, a culture is taken and, if positive, antibiotics are given as indicated.

Temperature elevation of more than 1° F. in the patient with tracheotomy means inadequate tracheobronchial care. Frequent suctioning and humidification will prevent or resolve this problem.

Delayed Postoperative Complications

Wound infection, fistula formation, and flap sloughing[12] are the major complications following neck dissection with or without resection of the primary lesion; they are relatively rare when a neck dissection alone is performed. In the combined procedure the reported incidence of these complications varies from 10 to 65 per cent. Patients who received 5,000 r or more of radiation therapy more than 8 weeks preceding surgery and who have large infected lesions are excellent candidates for one or more of the aforementioned complications. Prevention with compulsive adherence to the previously discussed principles is effective in most patients. When a fistula or skin slough occurs, however, procrastination and

watchful waiting serve no purpose. Immediate drainage, cleansing, and debridement will minimize the defect.

Infections respond best to drainage, frequent dressing changes, and irrigation of the wound with 5 per cent acetic acid. Although occasionally helpful, continuous irrigation of the infected wound immobilizes the patient and often is technically unsatisfactory. Despite all the previous precautions, some high risk patients still develop fistulas, flap necrosis, and exposure of the carotid artery. If the actual wall of the carotid artery develops an adherent, brown, dry crust, we believe that eventual rupture is almost a certainty. Thus, in the operating room after the patient's blood volume is corrected to normal and as oxygen is administered, the carotid artery is tied. Under such circumstances the morbidity and mortality (20 and 10 per cent, respectively) are significantly less than if one waits for the blow-out to occur and then ligates the artery.

Various procedures performed at the time of the original surgery have been described for prophylactic coverage and protection of the carotid artery should it become exposed. Dermal grafts,[10] a flap of prevertebral fascia, or a flap of the levator scapulae muscle[13] applied over the artery are the most frequently used techniques. Although each has its limitations and advantages, we presently favor covering the carotid artery with a superiorly based flap of the levator scapulae muscle. This muscle is readily available; the actual mobilization and suturing into place takes little time. On occasions when the carotid artery becomes exposed, the muscle usually remains viable and provides a fine bed for the split-thickness graft or regional flap used to close the defect.

Late Postoperative Complications

The very procedure of radical neck dissection may result in four types of complications: (1) neural, (2) sensory, (3) cosmetic, and (4) vascular. The neural deficits are the result of sectioning of the spinal accessory and cervical plexus nerves. The former results in motor denervation of the trapezius muscle, leading to inability to abduct the arm above the horizontal position, atrophy of the trapezius muscle and a "dropped shoulder." At least 30 per cent of patients develop these following radical neck dissection. The sensory deficits encountered as a result of sectioning the sensory routes of cervical nerves two, three, and four consist of anesthesia in the posterior aspects of the scalp, neck, and shoulder. A rare neural complication is gustatory sweating—thought to be due to abhorrant regeneration of autonomic nerve fibers. The cosmetic deficit results from the loss of the tissue bulk provided by the sternocleidomastoid muscle, producing a "flattened" appearance to that side of the neck. Persistent edema of the superior flap occasionally follows unilateral radical neck dissection. Initially headache, papilledema, facial cyanosis, and edema of the flap follow the second stage of a bilateral neck dissection. Although untold complications such as blindness have been reported, the other changes usually resolve in a relatively short period of time.

Because of these undesirable changes, some have modified the radical neck dissection in such a way as to eliminate the deficits—hopefully

without compromising the patient's chance for cure.[4] Preservation of the internal jugular vein on the second side of a bilateral dissection greatly diminishes the postoperative edema. Preservation of the external jugular vein on the second side – while time consuming and difficult – works as well as preservation of the internal jugular and theoretically is more acceptable to the cancer surgeon. A number of authors advocate sparing the spinal accessary nerve[11] or, if it is resected with the tumor, inserting a graft between the severed ends.[1] Except in patients undergoing neck dissection for papillary carcinoma of the thyroid gland, we do not believe in preservation of the eleventh nerve, sternocleidomastoid muscle, or the internal jugular vein. Preservation of the accessory nerve prevents complete dissection of the region of the subdigastric and superior deep cervical lymph nodes, both of which are frequently involved with metastatic carcinoma.

Emotional and psychological changes in some patients are perhaps the most difficult complications for the surgeon to handle. Frequently far too little attention is paid to this aspect of postoperative care. Support and encouragement by the patient's family, church, and surgeon are essential.

RESULTS

The type and location of the primary carcinoma and the status of the cervical nodes are the principal prognostic indicators. In general the small, well differentiated primary lesion with small cervical nodes, few in number, offers the best prognosis. Papillary carcinoma of the thyroid without clinical metastases to the lateral neck, for example, carries a much better prognosis than does anaplastic squamous cell carcinoma of the base of the tongue with cervical metastases. Patients with histologically positive nodes at initial surgery develop recurrences sooner and with greater frequency than patients in whom the nodes are histologically negative. Host resistance and the basic biological behavior of the cancer are important factors influencing survival. Both are very difficult to evaluate intelligently. In addition, neither necessarily remains stable throughout the course of the disease.

Approximately 33 per cent of all patients with histologic evidence of squamous cell metastases to the cervical lymph nodes are alive and well at 5 years. The salvage rate for patients who develop metastases to the cervical nodes on the second side of the neck without local recurrence and then undergo the second neck dissection is between 40 and 50 per cent. Only about 15 per cent of the patients are cured once they develop either a local recurrence at the site of primary resection or a recurrence in the neck that has undergone a previous dissection.

Some years ago distant metastases were reported in only 10 per cent of the patients with head and neck carcinoma. Recent studies in such patients indicate an increased incidence (up to 50 per cent) of distant metastases. Whether the change is secondary to patients living longer following more aggressive therapy, thereby allowing more time for devel-

opment of distant metastases, or whether the increased incidence is secondary to an alteration in the behavior of the lymph nodes following radiation therapy is not known. In rabbits, radiation therapy directed to regional lymph nodes somehow decreases the barrier function of the lymph nodes and allows for earlier passage of tumor cells through the lymph nodes to the next nodule station.

The mortality from radical neck dissection varies depending upon the extent of the cancer, general condition of the patient, prior radiation, and skill of the surgeon and anesthesiologist. The lowest mortality figure of 1 per cent occurs in non-irradiated patients who undergo radical neck dissection only. If the patient is irradiated first, the mortality increases to 6 per cent. When radical neck dissection is combined with excision of the primary lesion, 4 per cent of non-irradiated and 8 per cent of irradiated patients die in the postoperative period. For the entire group of patients undergoing radical neck dissection with or without resection of the primary lesion irrespective of whether or not radiation therapy was given preoperatively, the average mortality is 4 per cent.[8]

FOLLOW-UP

All patients are seen monthly for the first year, every 2 months for the second year and every 3 months for the third year. From then on and for the remainder of their lives, they are checked every six months. Patients are also repeatedly told that if a symptom such as dysphagia or pain or a sign such as swelling in the neck develops, they should not wait until their regular appointment, but should be seen immediately. At each visit the surgeon not only performs a careful examination of the head and neck to rule out recurrence of the original cancer, but also looks for the development of a new primary lesion. Patients who have had one carcinoma in the respiratory or upper alimentary tract are much more prone than the average patient to develop a second lesion in either of these areas. For the same reason all patients should be urged to stop smoking.

SUMMARY

The treatment of choice for patients with carcinoma of the lateral cervical region is the standard radical neck dissection. Cure rates not only depend on the site, size and type of the primary lesion, but are influenced as much or more by the degree of involvement of the cervical nodes. Little additional improvement can be anticipated in cure rates until tumor immunology, host-tumor relationships, and the basic biological behavior of tumors are better understood. The addition of either high or low dose preoperative radiation therapy may improve survival, but this has not been demonstrated conclusively. Life-long follow-up care that is both comprehensive and unrelenting is essential to detect recurrence or development of a second primary tumor.

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The Management of Complications in Head and Neck Surgery

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In surgical circles it is often stated that if one does the proper operation for the right patient, it is not necessary to be sophisticated in the postoperative management of the patient. There is some truth to this statement. It is often painfully clear that the patients of some surgeons seem to glide gracefully through their operative procedures and the patients of other surgeons are fraught with problems. Recognition of the potential complications of operative procedures in the treatment of head and neck problems is a prerequisite for the avoidance of complications. Although one goal of a good surgeon is to avoid complications, the ultimate challenge to a surgeon is the ability to skillfully manage a complication to a successful outcome. It is simpler to be objective about the complications encountered by another surgeon than those encountered by oneself. When one has not emotionally committed oneself to the patient in terms of the prior therapy, one can frequently be more objective in the successful management of the complication. It is for these reasons that avenues of consultation and rapport with fellow surgeons are necessary. There are few sins that are less pardonable than the refusal of a surgeon to recognize a complication, or attempts to cover up a complication to save face, to the detriment of the patient. The mature surgeon will recognize the problem early and will frequently seek consultation with his colleagues. Oftentimes the discussion does not yield new information, but does serve as a sounding board so the problem can be managed objectively.

PREOPERATIVE PLANNING

The preoperative evaluation of patients with head and neck cancer requires special considerations. Many of these patients have poor dentition. The correction of dental caries and proper dental hygiene will

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reduce the pathogenic flora in the mouth to minimize the risk of wound infections or dental complications.[30] Following previous operative procedures or x-ray therapy to the neck, patients have been discovered to be hypothyroid and hypoparathyroid. The symptoms of hypothyroidism are obvious in the young patient, but commonly the symptoms of lethargy, edema of the oral pharynx, hoarse speech, and constipation are attributed to the patient's tumor or previous therapy and not to the classic picture of myxedema in an adult. The poor response of the myxedematous patient to general anesthesia is well recognized[5, 11] and frequently will be erroneously attributed to "decreased blood volume," old age, or debilitation if the thyroid function is not evaluated.

Although a careful record of previous x-ray therapy may be available, the extent of tissue damage and vascular injury secondary to radiotherapy is often not apparent even to the experienced eye. Within the first month after treatment, inflammation and mucositis in the tissues is obvious and must be taken into account in the evaluation of the tumor and consideration of the potential problems of wound healing. Operative procedures undertaken subsequent to the initial phase of radiotherapy are fraught with problems since the degree of vascular injury is more extensive than the obvious tissue involvement in terms of skin erythema, atrophy, telangiectasia, and induration. For this reason the operative management must include consideration of the poor healing quality of irradiated tissues. Anticipation of complications in the previously irradiated tissue allows planning for available healthy tissues for pedicle flap replacement.

In the preoperative planning of a procedure, careful thought to the structures that will necessarily be removed and anticipation of the problems thereby introduced will allow the surgeon to consider most of the defects, and many of the complications can be avoided. Removal of the anterior body of the jaw with the consequent lack of support of the tongue should alert the surgeon to the need for reconstruction for tongue support or anticipation of a tracheostomy.[4, 20, 26] Removal of the base of the tongue or hypomandibular complex[15] and lack of protection of the larynx from secretions should alert the surgeon to the possibilities of airway obstruction or recurrent aspiration pneumonia. The need to divide the facial nerve in the course of the dissection may be anticipated and the use of a tarsorrhaphy to protect the cornea on the involved side can avoid subsequent keratitis and corneal ulceration with consequent loss of vision. Discussion of these anticipated problems with the patient leaves the surgeon in a position to follow whatever course he feels necessary and to avoid compromise in good judgment because some particular aspect had not previously been discussed with the patient.

Additional preoperative consideration should be given to the use of hypotensive anesthesia. It is advisable to seek anesthesia consultation prior to the anticipated operative procedure. Although the limitations are rapidly diminishing and many patients are considered candidates for hypotensive anesthesia, most anesthesiologists rightfully prefer to see the patient before the operation to evaluate his myocardial and cerebrovascular function prior to advising the use of such anesthesia. In those

procedures where uncontrolled blood loss can be anticipated, such as in heavily vascular tumors or in those lesions where bone resections include the maxilla, or where operative procedures in the pterygoid region are anticipated, hypotensive anesthesia has revolutionized the ability of the surgeon to carefully and without the pressures of rapid blood loss undertake a proper cancer ablative procedure.

OPERATIVE CONSIDERATIONS

Problems during surgery are common in head and neck surgery. Pressure on the carotid sinus at the bifurcation of the carotid artery can cause hypotension which in elderly people can be disastrous. Avoidance of pressure on the carotid bulb is the simplest means of avoiding this complication. If the patient has a sensitive carotid sinus and avoidance is not feasible, infiltration with 1 per cent xylocaine around the carotid bulb effectively reduces the carotid sinus response.

The assistant's heavy hand or large bulky clamps laying across the carotid artery can inadvertently occlude the internal carotid artery during the operative procedure and result in bizarre and sometimes disastrous complications. Elderly patients with arteriosclerotic disease of the carotid artery tolerate trauma to the artery poorly, and constant awareness of this vessel in the operative field must be maintained to avoid hemorrhage under an arteriosclerotic plaque or embolus. Although this is obvious at the time of dissection around the carotid artery, pressure or trauma to the vessel usually occurs while the attention is centered on a part of the dissection remote from the vessel.

To minimize venous pressure in the head and neck area, it is advisable to operate with the patient's back flexed approximately 30 to 40 degrees. In so doing the patient may have a negative venous pressure in the neck. A large vein may be opened and a significant amount of air may be sucked into the vein, most commonly the internal jugular vein. When this is inadvertently opened or opened without control, the rapid bleeding from the cephalad section of the vein is more impressive than the intake of air into the caudad end, and air embolization is a potentially serious problem. Prevention of this is critical, but when it is suspected because of sudden changes in blood pressure and possibly cardiac arrhythmias, the head of the table should be lowered immediately and the patient turned to his left side to allow the air to accumulate in the right atrium and right ventricle. If the patient does not respond to this maneuver, an immediate thoracotomy and direct aspiration of air from the right ventricle must be considered.[26]

Blood loss during operative procedures about the head and neck is usually well controlled, although there are several areas of resection which are hazardous. Dissection around the caudad portion of the internal jugular vein must be done carefully since the vein is quite thin walled and disappears quickly under the clavicle to join the innominate or subclavian vein. Tearing of this vein or of its juncture can result in serious hemorrhage and on occasions may necessitate removal of the

middle third of the clavicle in order to control the bleeding. Vigorous bleeding may occur from the internal jugular vein at its cephalad portion. In the properly carried out radical neck dissection for cancer, the internal jugular vein should be ligated as high as possible. It is in this high ligation that the risk of tearing the vein and uncontrollable hemorrhage occurs. Taking down the posterior belly of the digastric muscle allows access to the cephalad portion of the internal jugular vein in case damage to the vein occurs in the course of the usual dissection. When this tear extends up to the base of the skull, simply oversewing the vein may be required.

During major resections of the facial bones and resections involving the pterygoid area, there are many large vessels and diffuse bone bleeding which are inaccessible to hemostatic methods until the bone is actually removed. It is in these particular cases that hypotensive anesthesia has been most valuable. In an occasional situation in which the tumor or radiotherapy has distorted the anatomy, resection of the maxilla and pterygoid regions requires a rapid removal of the specimen in order to get at the internal maxillary artery directly.

It is well recognized that many patients will tolerate a loss of one carotid artery, but efforts on the part of vascular surgeons and neurosurgeons to predict which patients will tolerate ligation of the carotid artery have been frustrated. When the necessity to ligate this artery is known beforehand, it is sometimes feasible to use the Silverstone clamp gradually to occlude the vessel with the patient awake and thereby determine whether he will tolerate the loss of flow in that particular artery. However, the vast majority of these cases represent a complication of head and neck surgery or irradiation, and the opportunity to determine this beforehand has been lost. Since most of these patients have been irradiated and almost all of them have necrotic wounds or infection, the prospects of vascular grafts to replace the artery at the time of the hemorrhage are dim. However, there have been successful cases reported, and this alternative should be kept in mind. If, in the view of the surgeon, vascular replacement of the eroded vessel is not feasible, it is usually necessary to empirically ligate the carotid artery proximally and distally to the area of hemorrhage. The outcome depends upon the individual patient's ability to tolerate the low flow through the carotid artery, which is variable and unpredictable, and success depends upon maintaining an adequate blood pressure.[19] If the area of hemorrhage occurs below the bifurcation there may be sufficient flow above the ligation to retain adequate flow through the internal carotid artery and the prospects of survival are improved greatly. When the hemorrhage occurs at the bifurcation or in the course of the internal carotid artery, the prognosis is grim.

Ligation of both internal jugular veins at one operative procedure carries a mortality rate of 20 per cent[13, 14, 18, 27] because of inadequate venous drainage from the brain. If a bilateral neck dissection is necessary, it is preferable to modify one side of the dissection to leave one internal jugular vein intact. The alternative is to stage the dissections with a minimum of 1 month between dissections. Presumably this period of time allows the vertebral drainage to assume adequate proportions which then

allows removal of the remaining internal jugular vein.[10, 24] Massive edema, often persisting to a lesser degree, follows ligation of the internal jugular vein when the contralateral vein has been ligated or when previous irradiation to the contralateral neck has obstructed venous return.

Awareness of the thoracic duct lessens the likelihood of a lymph fistula postoperatively. On the left, the duct should be identified and ligated. Later examination of the lower neck for pooling of the clear or milky lymph further precludes overlooking a leaking duct and troublesome lymph accumulation or fistula postoperatively.

Involvement of peripheral nerves in radical neck dissection has been shown to be more extensive than usually appreciated.[29] In many operative procedures it is desirable to transect the spinal accessory nerve in the course of radical neck dissection.[22] However, in some procedures in the head and neck area where this is not indicated for purposes of proper cancer extirpation, inadvertent transection of this nerve does create a significant debility in that the nerve stimulates the sternocleidomastoid muscle and the trapezius muscle. The degree of disability varies from one patient to another, but one young man who was a barber by trade found that division of the spinal accessory nerve was a debilitating complication.

Unnecessary division of the branches of the facial nerve are unfortunate. There are many instances in cases of carcinoma of the parotid gland where the nerve must be divided. In these instances an elective tarsorrhaphy at the time of the operative procedure will minimize the risk of keratitis and corneal ulceration of the involved eye. Immediate repair of an inadvertently divided facial nerve is indicated and in young people the functional return is often excellent. When a section of the nerve is removed for tumor ablation, and when the proximal and distal portions are identified, a nerve graft should be carried out at the time of the original dissection.[32] Although there are no controlled series to indicate the superiority of this over secondary repair, the anatomic identification of the nerve branches is considerably easier at the time of the orginal dissection than secondarily. Successful nerve grafts have been done employing the sensory branches from the cervical plexus, the sural nerve, or other sensory nerves which are available without creating secondary debility. When direct repair of the nerve or nerve grafting is not feasible, it is often possible at the time of the initial procedure to dissect the anterior half of the masseter muscle as a pedicle based superiorly and transfer this to the orbicularis oris muscle directly to provide support of the left half of the face.

A common complication of surgery about the head and neck is the inadvertent injury to the ramus mandibularis of the facial nerve. This results in a disturbing flattening of the lower lip so that there is asymmetry especially on depression of the lower lip. Probably the simplest and most successful treatment of this injury, after a sufficient waiting period to satisfy oneself that the nerve will not recover, is to divide the contralateral ramus mandibularis and restore symmetry. If, however, the patient chooses not to accept this alternative, reasonable symmetry can be ac-

complished using a fascia lata graft between the inferior belly of the digastric muscle and the orbicularis muscle. Although this does not give perfect symmetry, it gives a natural muscle tone to the lower lip and corrects the elevated paralyzed half of the lip.

The hypoglossal nerve is a large structure which should be readily avoided in the course of a dissection. Injury to this nerve results in some loss of control of the tongue, but does not represent a catastrophe in itself. Transection or injury to the phrenic nerve results in a paralysis of the hemidiaphragm and in older patients can contribute to pulmonary complications. However, the long-term consequences of a phrenic nerve palsy are not usually significant. The recurrent laryngeal nerve is generally not injured except in patients undergoing thyroid surgery and the various maneuvers to avoid injury to this nerve and the careful examination and suspicion of injury to this nerve are well discussed in all of the literature concerning thyroid surgery.

When an intraoral procedure is carried out in continuity with a neck dissection, integrity of the mucosal closure is critical. Tension of the wound, devascularization of the tissues from injudicious dissection, use of previously irradiated tissues, and poor handling of tissues can result in breakdown of the mucosal closure. When the breakdown opens into a neck dissection area with access to the carotid vessels, the results can be disastrous. Removal of more mucosa than originally anticipated, extension of tumor, miscalculation of the extent of the wound defect, or encountering borderline tissue secondary to irradiation are common problems in major head and neck procedures. The surgeon operating in the head and neck should have at his disposal various pedicle flaps which would be immediately available to him during the course of the operative procedure.[6, 9, 32, 33] To limit the operative resection of the tumor or to compromise the wound closure because of the lack of ability to move in healthy tissues limits the surgeon's ability to handle the problems competently. When the breakdown of the mucosal closure occurs, immediate repair of the wound is usually indicated. Most often this necessitates the use of nearby pedicle flap tissue. Bleeding of the carotid artery associated with a salivary fluid is disastrous. In some instances the skin wound can be opened and the fistula can be directed from the hypopharynx to the skin; the fistula may thereby be controlled until an opportune time for closure.

A lymph fistula more commonly occurs on the left than on the right because of the thoracic duct, but there are significant lymphatic ducts on the right in most patients, and it is not an unknown complication. Avoidance of this complication at the time of the procedure by awareness of the thoracic duct posterior to the jugular vein and by looking for the appearance of clear lymph in the wound is preferable. When a fistula appears early in the postoperative period and is obviously vigorous, serious consideration should be given to opening the wound and identifying the lymphatic trunk. If, however, this was done at the operative procedure and there are accessory lymphatics which are leaking, conservative management is usually successful. Small leaks with some appearance of lymphatic fluid in the neck, which can be controlled with daily aspiration and

pressure dressings, usually cease after a reasonable period of time (5 to 10 days). Large leaks can introduce fluid and electrolyte problems temporarily.[23]

POSTOPERATIVE COMPLICATIONS

Postoperative hemorrhage following head and neck surgery can be dramatic. The two most ominous hemorrhagic problems occur from the carotid artery and from the innominate artery.[3, 13, 19, 31] Most exsanguinating episodes of hemorrhage from a carotid artery are seen in patients who have had previous radiotherapy and in whom wound breakdown has occurred. Efforts to minimize the risk of postoperative hemorrhage from the carotid artery have included the use of the levator scapula, posterior scalene, or sternohyoid neck muscle[16, 25, 28] elevated as a pedicle after transection distally, or free dermal grafts.[8] When the tissues overlying the artery are heavily irradiated or contaminated, it is frequently advisable to consider preoperatively the use of a vascularized pedicle flap of skin which will provide healthy coverage over the vessel. Hemorrhage may occur from erosion of the innominate artery either by a low tracheostomy tube or a fistula between the innominate artery and the trachea.[3, 31] Unless recognized immediately, the innominate artery-trachea fistula is usually fatal. If recognized, ligation of the innominate artery may be carried out with impunity. Most patients who develop significant bleeding from either of these two major sites exhibit early signs that herald the ominous event. It is critical for the doctors and nurses caring for these patients to recognize that any oozing occurring in the postoperative period, especially after the first 24 hours, should be considered bleeding from one of these major vessels and steps taken to prepare for major resuscitative procedures.

Postoperative hemorrhage in head and neck procedures can result from a number of situations. The use of hypotensive anesthesia during the operative procedure may delude the surgeon into believing that adequate hemostasis has been established if the blood pressure has not returned to normal prior to closing the wounds. Early in the experience with hypotensive anesthesia it was believed that if it were maintained throughout the procedure and the wounds closed, the pressure dressings and wound closure would avoid any bleeding when the blood pressure returned to normal. Experience over several years with hypotensive anesthesia has led us to return the blood pressure to normal after the ablative portion of the procedure is completed. This need not be done with drugs, but simply allow the patient's blood pressure to drift back to normal. Extubation of the trachea and frequent tracheal toilet result in explosive episodes of increased venous pressure which can result in venous bleeding in the head and neck region either from sutures being inadequately secure or from the use of electric cautery. Smooth extubation is a critical part of the operative procedure and the surgeon should make the anesthesiologist aware of this requisite.

A tracheostomy is frequently a life-saving device and is particularly

critical in many of the head and neck cancer problems. On the other hand, a tracheostomy is not to be taken lightly. Today most surgeons would agree that tracheostomy should be done in the operating room and whenever feasible as an elective procedure rather than considered to be a bedside procedure to be used in dire emergencies. Large veins around the trachea in older patients with increased venous pressure can bleed dramatically, and vascular anomalies over the trachea are possible.[1]

In children the apex of the lung is high and the incidence of pneumothorax following a tracheostomy is significant. Careful consultation of the chest is indicated immediately after tracheostomy and chest x-ray as soon as feasible is desirable. It has been recognized for some time that there is a peculiar syndrome of sudden death following tracheostomy attributed to diminution of a stimulus for breathing which occurs in the immediate posttracheostomy period.[2] For this reason all patients who have had tracheostomies should be carefully observed in an intensive care unit or recovery room.

The problems of laryngotracheal obstruction complicating tracheostomy have been thoroughly examined and discussed by Harley.[12] Placement of the tracheostomy below the second tracheal ring, avoidance of prolonged use of cuffed endotracheal tubes, the use of small tubes, and minimization of frequent tube changes are important in reducing the chances of laryngotracheal obstruction.

The postoperative management of the tracheostomy patient is critical. The use of the new synthetic tubes with cuffs have minimized the incidence of mucous plugs of the tracheostomy tube per se. The tracheostomy tube in a heavy individual with a thick neck can become displaced and actually cause obstruction of the airway. To minimize the likelihood of displacement of the tracheostomy tube, heavy nylon sutures may be placed through the tracheal cartilage and brought out to tie to the tracheostomy tube or to be left long in case the tube is displaced in the first few days postoperatively and traction on the two sutures on the tracheal cartilages will allow replacement immediately. Usually umbilical tape is used to tie the tracheostomy tube around the neck, but efforts to prevent this from being tight can result in its being loose and allowing displacement of the tube. The umbilical tape tie should be checked at regular intervals to ascertain that it is performing its purpose. In many instances it is desirable to actually suture the tracheostomy tube to the skin around the tracheostomy stoma to minimize the likelihood of displacement. Since patients with the tracheostomy are unable to call for help, they should never be placed alone in a room. When proper nursing supervision is not possible, it is frequently feasible to place the patient in a semiprivate room with a cooperative patient who will serve to call for help if the need arises. Frequent cultures of aspirate in patients with tracheostomies are indicated to detect early pneumonias. Pseudomonas infections are seen in patients on respirators. The respiratory care of the tracheostomy patient is a challenge to the surgeon and has caused most surgeons to consider carefully the indications for tracheostomy in the first place and to remove the tracheostomy as soon as it is safe. An excellent discussion of those factors is found in *Respiratory Care*.[2]

Upper airway obstruction is the major concern for patients in the immediate postoperative period following surgery of the head and neck region. Edema of the epigolottis and larynx are common following irradiation to the neck region and this can result in airway obstruction with the added insult of intubation or minimal operative procedures. Diagnostic endoscopy and biopsies of laryngeal lesions in patients with previous irradiation can precipitate upper airway obstruction, and these procedures should always be carried out with the patient hospitalized and under careful scrutiny.

Resection of the anterior body of the mandible often results in lack of support of the tongue, and mechanical airway obstruction can ensue. Prevention of this problem with immediate support of the mandible either with bone grafts or artificial splinting can in most instances minimize this danger. In anticipation of the mechanical problems associated with major resections of the oral cavity, and on considering the transient lack of ability to handle secretions with edema or partial palsy of the tongue, many surgeons prefer to include a tracheostomy as a part of the operative procedure. Judgment on the advisability of the tracheostomy is entirely a matter of experience. When the tongue has been partially resected and mobility of the tongue is limited because of suturing or because of extensive edema in the immediate postoperative period, the patient's ability to swallow secretions is handicapped and aspiration of saliva can be a hazard. Postoperative dysphagia consequent to oropharyngeal resections has been managed successfully by sectioning the cricopharyngeus muscle.[4, 17]

Few non-fatal complications are more disconcerting than uncontrolled drooling after head and neck operations. Restoration of the anterior and lateral gutters along the floor of the mouth and mandibular reconstruction are the most effective preventive measures. A bridge of scar tissue between the posterolateral tongue and the tonsillar fossa is frequently the obstruction factor in preventing adequate drainage of saliva into the hypopharynx and this should be corrected when feasible. When all other measures fail, removal of the remaining submaxillary glands and redirection of the parotid ducts posteriorly to the anterior tonsillar pillar are effective measures to eliminate or noticeably reduce drooling.[7]

Postoperative parotitis used to be a serious complication in patients following head and neck operations. Staphylococcal suppurative parotitis had a high mortality rate. With effective antibiotics, early institution of therapy when parotitis is diagnosed has essentially eliminated this once serious problem.

After a parotidectomy, collection of parotid fluid and parotid cutaneous fistula are not rare complications. The majority of these fistulas will heal spontaneously and no therapy will be necessary. Gustatory sweating in the temporal and parotid region (Frey syndrome) occurs in a significant number of patients following parotidectomy. The majority of these patients will find this annoying syndrome to be self-limiting. Efforts at correcting the sweating in patients in whom this has persisted have led to many ingenious procedures related to the ganglia, but the results have been frustrating.

In the absence of previous irradiation, wounds in the oral cavity, hypopharynx, and the neck region heal remarkably well. Persistent fistulas are the result of previous irradiation, tumor, or extensive tissue loss as a result of ischemia or massive infection. In all of these instances removal of the irradiated tissue, tumor, or scar tissue in the region of the fistula and closure with additional healthy pedicle tissue are the most direct means of closure.[6, 9, 32, 33] Efforts to pull the tissues together with large sutures are rarely successful and often simply prolong the complication.

Radiation necrosis of the mandible may be low grade, and superimposed operative procedures may precipitate sequestration of the bone. Chronic draining sinuses and unremitting pain are the unpleasant accompaniment of this complication. Invariably resection of the involved mandible is indicated, and usually the involvement is more extensive than is apparent preoperatively.[21]

SUMMARY

Recognition of the potential complications during or subsequent to operative procedures about the head and neck allows the surgeon to avoid these problems or to recognize them in their early stages. Most fatalities directly related to the operative management of these patients occur from airway obstruction or from exsanguinating hemorrhage. Although patients with extensive tumor and extensive irradiation changes about the head and neck may necessarily succumb from these two complications, fatality from airway obstruction or exsanguinating hemorrhage is avoidable in the patient with early disease.

Careful attention to the anatomic structures in the head and neck region allows definitive decisions as to the transection of nerves, avoidance of lymph fistulas, and proper cancer ablative procedures. Reconstructive concepts which permit removal of irradiated or infected tissues minimize the likelihood of wound breakdown. Preoperative planning of functional problems permits control and reconstruction to minimize the morbidity of postoperative dysphagia, drooling, and cosmetic deformity.

Any surgeon undertaking operative procedures about the head and neck must be thoroughly familiar with all of the potential complications and the concepts which will allow him to avoid and manage these problems with a degree of sophistication in keeping with the current status of modern surgery.

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Surgical Management of Pain in Cancer of the Head and Neck

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Cancer of the head and neck is distressing to the patient not only because of pain and difficulties with swallowing and breathing, but because he may also need to contend with the disfigurements of ablative surgery, the embarrassment of a malodorous cavity, and the not infrequent dryness and burning sensation that follow radiotherapy. Though he may complain most loudly of his pain, this may, in fact, merely represent the focal point of his distress which may not be greatly altered by mere elimination of the pain component.

The first matter, therefore, in treating a patient with pain from cancer in this area is to understand his total problems as completely as possible and deal with all of them, utilizing such help as gastrostomy and tracheostomy will provide at the appropriate time. The next is to plan an adequate medical regimen for the control of pain and anxiety. This will vary according to the psychological needs of the patient. The basis is a generous use of the non prescription and simple analgesics. It is often advisable to prescribe a small amount every hour rather than a large amount every 3 hours. This is partly because a better blood level can be obtained, but mainly for the psychological reason that help is always at hand. The next important medicine, and sometimes the more important, is an adequate dose of potent tranquilizer. It should be recalled that chlorpromazine (Thorazine) in a dose of between 400 and 500 mg. daily will do almost anything that frontal lobotomy can do and can do it in a reversible manner. The surgeon, therefore, has at his disposal a simple reversible pharmacologic lobotomy. A dose of 200 mg. of chlorpromazine is often adequate. Patients will frequently complain of being lethargic on higher doses, and a level at which lethargy and anxiety are titrated is worked out. In some instances, infiltrative cancer of this area produces a stabbing pain which slightly resembles trigeminal neuralgia, rather than the more common deep, dull pain. These stabbing pains may respond to

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carbamazepine (Tegretol), 200 mg. four times daily. Carbamazepine may produce nausea and ataxia, and the patient has to be watched for white blood cell and liver toxicity, but in the appropriate case it is a very valuable drug.

The simple narcotics, such as codeine and oxycodone (Percodan) have limited usefulness. With increasing doses they soon cause anorexia and constipation and initiate a downhill course. It is certainly appropriate at this stage to consider initiation or renewal of a course of chemotherapy or radiotherapy if possible. It may be that chemotherapy does not result in a significant number of cancer cures in this area, but it does appear to have some ability to suppress pain with no risk of causing pain. The same cannot always be said of radiotherapy.

When narcotics seem indicated the drug of choice is methadone. It has a significant amount of the anxiety-relieving and pain-relieving qualities of morphine, without the concomitant depression, depersonalization, and catabolism of morphine. It can be provided simply in oral form, taken with fluids, or administered through a gastrostomy. As a general rule, medication by injection, which makes a patient dependent on hospital or physician, should be avoided.

ANATOMIC CONSIDERATIONS

Before commencing any form of narcotic therapy it is advisable to consider what surgical methods have to offer. The area is a difficult one anatomically, served by almost innumerable branches of the fifth, seventh, ninth, and tenth cranial nerves, as well as by the cervical plexus. The upper nasal cavity is supplied by two branches of the anterior ethmoidal nerve (a branch of the ophthalmic division of the fifth) which, traversing the orbit, finally emerges on the surface as the external nasal nerve between the nasal bone and the upper nasal cartilage. This branch contributes to the vestibule. The anterior ethmoidal and its companion (when present), the posterior ethmoid, supply the ethmoid and sphenoidal sinuses. From the infraorbital branch of the maxillary, the posterior dental nerve supplies the molars and the lining of the maxilla, the middle superior dental supplies the premolars, while the anterior superior dental supplies the incisors and canines and contributes to the floor and lateral walls of the anterior nose. Further nasal branches of the infraorbital nerve supply the anterior inferior septum. The short sphenopalatine nerves supply the upper back part of the nose and the posterior ethmoids. The long sphenopalatine contributes some sensation to the septum, but penetrates the hard palate anteriorly through the incisive canal to supply the anterior portion of the roof of the mouth. The posterior portion of the hard palate together with the posterior floor of the nose and some of the soft palate are supplied by the greater palatine nerve. The lesser palatine nerves deal mainly with the uvula, tonsil, and soft palate, while a special pharyngeal nerve supplies the nasopharynx. The contributions of the mandibular nerve are the buccal to the buccal mucosa, including the buccal portion of the posterior gum, the inferior dental to the teeth, and the lingual to the anterior two thirds of the tongue and adjoining mucosa. In

addition, branches of the superficial temporal nerve supply the external auditory meatus.

The facial nerve is not usually considered to be a sensory nerve, but does, in fact, have a superficial skin component as shown by the fact that blisters occur in the external auditory meatus when affected by herpes. Its better recognized sensory component, the chorda tympani, leaves it in the temporal bone and joins up with the lingual branch of the mandibular fifth to supply taste to the anterior two thirds of the tongue. The ninth supplies the tympanic area, the pharynx, and the tonsil by designated branches and supplies both common and taste sensation to the posterior third of the tongue.

From the tenth, the internal division of the superior laryngeal nerve divides into an upper and a lower branch. The upper gives twigs to the pharynx, the epiglottis, the vallecula, and the vestibule of the larynx. The lower supplies the pyriform fossa and the back of the aryepiglottic fold. The recurrent laryngeal supplies the larynx below the vocal folds. A tiny branch of the vagus makes its way to the posterior auditory meatus. The cervical plexus supplies the neck, most of the ear, and the skin over the posterior inferior corner of the lower jaw.

Thus it is seen that the nose is innervated from both the first and second divisions of the fifth, and the tongue by the third division of the fifth and the ninth, and most posteriorly in the valleculae by the tenth. The pharynx is supplied by the second division of the fifth and by the ninth. The ear has contributions from the fifth, seventh, ninth, and tenth, and from the cervical plexus.

NERVE BLOCK

Regional blocking by alcohol thus has serious technical problems. Blocking of the first division of the fifth cranial nerve is impossible because it is technically inaccessible. In addition, even if available it would be undesirable because it would destroy corneal sensation, which could lead to corneal ulceration. Blocking of the second division is technically possible in the sphenopalatine fossa and is useful for maxillary carcinomas. A needle can be inserted into the infraorbital foramen and may reach far enough back to involve the anterior and middle superior dentals, but since it will not get the posterior dental or the sphenopalatine or palatine nerves, it is not of much use. If a second division block should be carried out, it is best done in the sphenopalatine fossa by a needle inserted anteriorly and superiorly through the coronoid notch of the mandible (Fig. 1). The lingual nerve can be reached easily from inside the mouth at a point opposite the second molar, but, again, if a block is desired it is usually best accomplished by involving the total third division at the foramen ovale. For this purpose, the needle may be inserted by the lateral approach through the coronoid notch or by a more anterior approach from a skin point 1 cm. above and behind the angle of the mouth. For pains likely to last no more than 6 months, a block of the nerve at the foramen ovale is acceptable. For longer duration, repeated

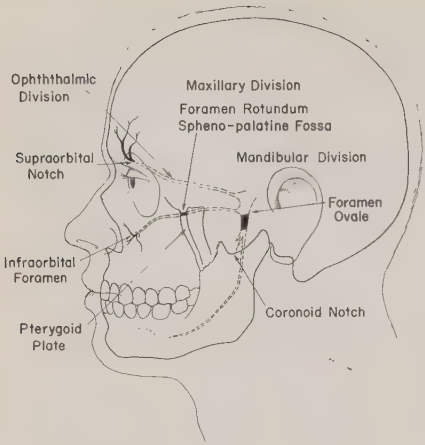

Figure 1. Diagram to illustrate access to the branches of the trigeminal nerve. (The coronoid process has been omitted to show the pterygoid plate.) The second and third divisions are approached through the coronoid notch. The ganglion which lies inside the skull deep to the foramen ovale may be reached through the coronoid notch, but an approach medial to the coronoid process from a point one centimeter lateral to and above the angle of the mouth is preferable.

injections are possible. However, a single selective block within the ganglion is more desirable because destruction of ganglion cells persists indefinitely. This injection is technically more difficult. Occasionally, if the function of the ninth and tenth nerves has already been largely destroyed by tumor, one may block residual sensation in these at the jugular foramen. It is not possible to selectively block either the ninth or the tenth nerve or block sensation while preserving motor function. An all or nothing result must, therefore, be accepted.

NERVE DIVISION

For pains confined to the second and third divisions of the fifth nerve, alcohol block is usually satisfactory, but if the technical skill is not available or if the patient will not tolerate the significant pain involved in such blocks, then selective division of the fifth nerve in the middle fossa is a standard and relatively simple neurosurgical procedure with a low morbidity and virtually no mortality rate.

MULTIPLE CRANIAL NERVE SECTION

With widespread involvement of the lower cranial nerves multiple divisions within the posterior fossa on one side are technically possible, but there are problems. The fifth is at a great depth. The intermedius branch of the seventh is difficult to locate without damaging the eighth, thus producing hearing loss. The point of demarcation between the upper fibers of the tenth and the lower fibers, which provide sensation and movement to the larynx, is a difficult and arbitrary one. Thus, the operation is technically difficult and subject to technical failures in its effort to relieve the desired amount of pain. In addition, it carries the undesired effect of removing touch sensation from a wide area as well as pain. It has

the morbidity of undesired damage to adjoining nerve fibers and, in these debilitated persons with long-standing cancer, a significant mortality factor. It has not found wide acceptance; though, for the properly selected patient, when performed with the proper amount of skill, it can relieve pain.

MEDULLARY TRACTOTOMY

A technically simpler procedure than the division of the sensory roots of four cranial nerves is an incision into the descending pain tract of the lower medulla where these sensory fibers converge and descend to connect with the second order of neurones (nucleus caudalis) before ascending again in the brain stem. The procedure has another advantage over multiple root section in that touch fibers have already separated, and the incision involves no loss of touch sensation. It has, however, a very major disadvantage. The descending tract, so clearly seen in anatomic diagrams, and even in formalin-fixed specimens, is not visible on the surface of the living medulla. Its approximate anatomical position is known, but an incision too medial will result in proprioceptive loss in the ipsilateral arm with resulting clumsiness, and one too laterally in pain loss of the contralateral side. One which risks neither medial nor lateral extension may be too narrow and thus fail to eliminate the necessary amount of oronasopharyngeal pain fibers (Fig. 2).

This problem of localization has now been solved by studies carried out in the neurosurgical laboratory of the University of Chicago in recent years. For a long time neurophysiologists have been familiar with the evoked potential technics in animals in which central nervous system areas which responded to peripheral stimulation were located and mapped. It was thus a comparatively simple matter to adapt these methods to the human. At the beginning of the operation, percutaneous stimulating electrodes are applied to the frontal, infraorbital and submental nerves and to the median nerve (Fig. 3). Pick-up electrodes are then applied to the exposed medulla, and the location of the descending tract of the fifth and the proprioceptive tract of the arm are mapped with absolute precision, using an oscilloscope. The order of fibers from lateral to medial is ophthalmic, maxillary, and mandibular, though only the

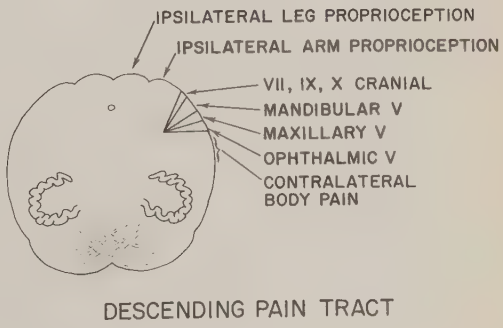

Figure 2. Map of neural function of the medulla.

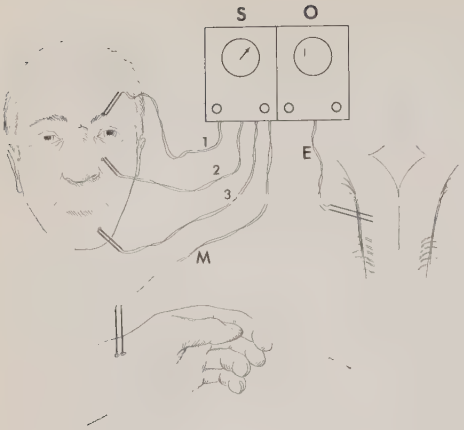

Figure 3. Diagram to illustrate points of peripheral nerve stimulations which will map the posterior surface of the medulla by evoked potential recording. S, stimulator; O, oscilloscope.

ophthalmic reaches the lowest level. Next come the seventh, ninth, and tenth fibers, in that order. Medial to these are the proprioceptive fibers from the arm, and finally the proprioceptive fibers from the leg, which adjoin the midline. Thus, a tractotomy confined to the descending, pain conducting tracts can be done with absolute accuracy. This technic of maximizing the precision eliminates most of the complications and extends the usefulness of medullary tractotomy considerably. Nevertheless, it does involve a modified posterior fossa exposure, and even the most minimal posterior fossa operation is not easily tolerated in those terminally ill patients. They may require 3 to 6 weeks to regain their preoperative degree of well being, making the procedure only really useful to those who have more than 3 months to live. In the immediate postoperative period there is a danger of respiratory failure if edema from the tractotomy site in the medulla should spread into the adjacent respiratory center. Such failure can be prevented by the use of preoperative and postoperative dexamethasone (Decadron) to minimize cerebral edema, by frequent PaO_2 determinations to monitor the onset and progress of respiratory difficulty, and by the use of temporary respiratory support in those instances when it is needed. The main decision of the surgeon is whether his terminally ill patient will support a major operation. If his judgment is good, there is virtually no mortality. If there is a serious doubt, it is best to initiate or continue narcotics. In order to afford maximum relief, surgery should be offered at an early stage of a predictably long and difficult illness, rather than at a stage when narcotics have lost their usefulness.

CORDOTOMY

Head and neck cancer rarely infiltrate the brachial plexus. In those rare cases of involvement, percutaneous cordotomy offers an effective form of relief.

VERTEBRAL FUSION

The pain of some very slowly growing tumors which infiltrate the cervical vertebrae, such as chordoma, cancer of the breast, and cancer of the thyroid, can sometimes be relieved by cervical fusion, with or without excision of the involved bone.

INDICATIONS

In general, epithelial cancers of the anterior mouth and tongue may give pain and discomfort at an early stage. They respond fairly well to curative treatment and do not present a major source of intractable pain. When they do, their regional site makes them well suited to alcohol block of the mandibular division of the fifth, or even surgical division of that branch. Carcinoma of the posterior tongue and pharynx rarely presents with early pain and reaches the neurosurgeon at a late stage of deep involvement, following multiple surgical and radiotherapeutic endeavors. If unilateral, and the patient has a reasonable life expectancy, then a medullary tractotomy is indicated. Most, however, have a short life expectancy at this time. If the infiltration and pain are bilateral, as they often are, then surgery is not useful. Although it is technically possible to do a bilateral medullary tractotomy, these patients are simply not well enough to tolerate two major staged posterior fossa operations. Alcohol block of the fifth is sometimes worth trying, but it is usually disappointing because infiltration and pain here spread to a much wider area.

Carcinoma of the maxilla is regionally suited to alcohol block of the maxillary branch of the fifth, but when confined to this territory is usually controlled by surgery and radiation. As a rule, it does not present as intractable pain until it has extended to a wide territory and is then most suited to medullary tractotomy. Nasopharyngeal carcinomas often infiltrate through the base of the skull and cause multiple cranial nerve palsies, but the resulting symptoms are better cared for by radiotherapy than by cranial nerve section or even tractotomy. The infiltration is, in fact, frequently bilateral. Parotid carcinoma is a deeply infiltrative tumor, sometimes of rather protracted duration, and is one of those best suited to medullary tractotomy. Laryngeal cancer, usually controlled by therapeutic means, is rarely so extensive as to require palliative surgical control of pain. If the infiltration involves the cervical plexus, then a dorsal root rhizotomy will eliminate that component. If it involves primarily the territory of the ninth and tenth cranial nerves, and the patient has already lost function of the ninth and tenth cranial nerves (has a tracheostomy and a gastrostomy), an alcohol block of the jugular foramen is occasionally indicated. Occasionally, the territorial infiltration indicates a full medullary tractotomy. As these patients have often a prolonged life span, this pain can be a very distressing one.

Those somewhat rare orbital tumors which destroy the orbital contents are helped by alcohol injection of the trigeminal ganglion. With particular skill, it may be possible to inject the first and second divisions selectively while sparing the third.

Following extensive infiltration, radiotherapy, and ablative surgery, the patient may have a deep facial cavity with an extremely tender stump of a trigeminal branch in its base. This can provide one of the most agonizing of all cancer pains. It can be managed by direct infiltration of the stump with alcohol, preferably at the site of its existing foramen, which is one of the simplest of all surgical procedures. It should be remembered that, as with all alcohol blocks, the nerve should be first infiltrated with procaine, as alcohol, on the unanesthetized nerves, gives rise to a most distressing burning pain.

SUMMARY

Surgical interruption of pain-conducting nerves in the treatment of cancer of the head and neck is a small, but very useful, aspect of total management. In general, it should be considered before resorting to narcotics. The major problem is that this area is a meeting ground of pain conducting components of the fifth, seventh, ninth, and tenth cranial nerves and the upper branches of the cervical plexus. Thus, alcohol block or division of single nerve branches has limited usefulness. Medullary tractotomy, which takes care of the regional area, has been greatly improved in accuracy by the adoption of the evoked potential localization technique, but it remains a very major operation for a terminally ill patient. It should thus be advised at the early stage of a predictably long and painful illness, rather than at an end point when narcotic medication no longer gives relief. At this early stage, it is virtually devoid of mortality or morbidity.

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Hyperparathyroidism Today

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The remarkable increase in the incidence of hyperparathyroidism throughout the United States and in a number of European countries, such as Sweden, is the most significant recent development in this disease. Superimposed on this abundance of patients with "primary hyperparathyroidism" diagnosed in the early stages of the disease, is the ever increasing number of patients on chronic renal dialysis who live long enough to develop serious complications and symptoms from what has been designated as "secondary hyperparathyroidism."

The time-honored description of "primary hyperparathyroidism," consisting of a mixture of bone disease, renal stone disease, gastrointestinal symptoms, and the finding of a single adenoma with three atrophic glands at exploration, is losing its validity: it is becoming increasingly clear that hyperparathyroidism is a nonspecific response to a variety of stimuli, where one pair or both pairs of parathyroids hypertrophy and undergo hyperplastic changes. With the passage of years, one or more glands may reach very large dimensions while the others may remain hyperplastic, normal, or may even atrophy.

An intriguing clinical classification of the various forms of hyperparathyroidism has been suggested (Table 1).[3] Eventually we may find that "hyperparathyroidism" consists of several separate entities defined according to specific etiologies. One may go a step further and predict, on the basis of some preliminary observations, that the superior glands may be involved in one clinical form (renal calcinosis) and the inferior in another (bone resorption).

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Table 1. *Classification of Hyperparathyroidism*

FORMS	COURSE	CALCEMIA	GLAND SIZE
Stone	Slow	Mild	Small
Bone	Rapid	Marked	Large
Chemical	Unknown	Moderate	No Pattern

STIMULI TO THE PARATHYROIDS

Calcium deficiency and/or the intake of an excess of phosphates are well known methods of producing parathyroid hyperplasia in laboratory animals. Recently a number of specific stimuli to the parathyroids and clinical situations in which these glands become hyperplastic and hypertrophic have been identified. The specific agents known to stimulate the parathyroids are thyrocalcitonin, ACTH, glucagon, propylthiouracil, and thiazide diuretics. Clinical situations in which parathyroid hyperplasia and hypertrophy have been observed include multiple pregnancies, chronic pancreatitis, malabsorption syndromes, waxing and waning metastatic breast cancer, chronic renal insufficiency, Sipple's syndrome, and pseudohyperparathyroidism.

In general any recurring stress to calcium metabolism such as multiple pregnancies, waxing and waning cancer of the breast metastatic to bones, and gastrointestinal malabsorption, especially when treated with steroids which further reduce calcium absorption, all can be conditions etiologically related to the development of hyperparathyroidism in patients with genetic predisposition.

The noted increase in incidence is due in part to the screening of large segments of the population for hypercalcemia; this has been facilitated by the introduction of automation in the clinical laboratory. However, there must be other factors which are responsible for an actual increase, such as the widespread usage of thiazide diuretics.[5, 6] The large numbers of patients on chronic renal dialysis have already been mentioned.

Finally, endocrine imbalances that produce an increase in circulating thyrocalcitonin or glucagon, two hypocalcemic agents, lead to parathyroid hypertrophy and hyperplasia.

PATHOLOGY

With the advent of near-total ($3\frac{1}{2}$) parathyroidectomy for the treatment of this disease, pathologists have had the unusual opportunity to study the histological changes in all four glands in these patients: their findings do not correspond to the classic descriptions and classifications that still appear in current textbooks. The entire spectrum of "adenoma," hyperplasia, transitional cells, and normal histology may present not only in a single patient, but in some instances in a single gland.[9] In addition, the so-called resting or dormant oxyphile cell is bursting with mi-

tochondria, a morphological characteristic incompatible with inactivity. The hyperplastic changes in the same patient and in a single gland may involve all three varieties of parathyroid cellular components. It is for these reasons that until a better understanding of this disease is achieved the pathological classification will remain very general: multiglandular disease, single adenoma, or carcinoma.

The first category, multiglandular disease, comprises the largest number of patients (65 per cent in our series of 84 patients), in which more than one gland displays hyperplastic and/or hypertrophic changes; the second consists of patients in whom only one gland is enlarged and the other three are normal or atrophic grossly and histologically (33 per cent); and the third and rare category comprises patients with carcinoma (2 per cent in our series); these are generally patients with demonstrated distant metastases.

DIAGNOSIS

In most medical centers the diagnosis of hyperparathyroidism still hinges on the accurate determination of serum total calcium, ionized calcium, and phosphorus.

The most accurate and practical measurement of parathyroid activity available today is the determination of ionized calcium.[4]

The radioimmunoassay of circulating parathormone, which is under development, will eventually provide a more specific tool for the assessment of parathyroid function and may improve diagnostic accuracy. However, the routine availability of this immunoassay is several years off and certainly will not replace the diagnostic tests used currently. Furthermore, it has been shown that parathormone levels are very sensitive to a moderate degree of renal functional impairment[7] and therefore the significance of elevated serum parathormone in a normocalcemic patient may be difficult to interpret. At present it would be very difficult to make a case for neck exploration in a normocalcemic patient whose serum parathormone may be elevated and who does not demonstrate any complication of hyperparathyroidism. Today, however, most endocrine surgeons and endocrinologists would not hesitate to recommend exploration in such a patient if the serum calcium were 12 or 13 mg. per 100 ml. The question of normocalcemic hyperparathyroidism, generally diagnosed in patients with renal calcareous disease, is a difficult one. In many instances such patients have "borderline" elevations of their serum calcium or they have not been evaluated over a sufficiently long period of time, especially through the spring and summer seasons. Sooner or later the patients with true hyperparathyroidism will demonstrate hypercalcemia. The others probably have idiopathic hypercalciuria, a disease of unknown etiology.

The assessment of the mineral content of bone has proven useful in differentiating these two diseases:[1] patients with idiopathic hypercalciuria have normal bone densities, in spite of the restriction of calcium intake for years, whereas generally in hyperparathyroidism there is a significant decrease in bone density, which indicates that the patient has

been in a state of negative calcium balance for a long time. For the same reasons, the determination of bone density has been useful in the differentiation of patients who have occult nonparathyroid malignancies that secrete parathormone-like substances or vitamin D-like sterols (pseudohyperparathyroidism), from the ones with true hyperparathyroidism. Usually, in pseudohyperparathyroidism, the abnormal state of negative calcium balance has not been of sufficient duration to produce any detectable change in bone density, even though this is a far more sensitive determination of demineralization than conventional radiologic techniques.

Finally, the various techniques of parathyroid localization utilizing scans, angiograms, massage and immunoassay of venous effluents from neck veins for parathormone, and barium swallows are all of dubious value, except in patients who have had previous unsuccessful explorations. The following illustrative example is a case in point:

Case 1

This 56 year old housewife was admitted in November of 1969 with a long history of epigastric distress, nausea, and hypertension treated with thiazides and reserpine. Hypercalcemia and hypophosphatemia had been noted in another hospital 2 weeks earlier. The past history was unremarkable. The patient's weight had remained stable. She had been prone to episodes of depression and had received a variety of medications to combat this. Physical examination was essentially normal; the blood pressure was 115 mm. Hg systolic, 85 diastolic. Serum calcium levels ranged between 12.0 and 15.0; phosphorus between 1.4 and 2.5. Bone density was markedly diminished.

At exploration the left superior, the left inferior, and right inferior glands were excised in toto; they weighed, respectively, 40 mg., 25 mg., and 40 mg. Approximately 10 mg. of what was thought to be the right superior gland was excised. On frozen section all four glands were interpreted as parathyroids. The left superior and the left inferior were 90 per cent cellular and considered hyperplastic. The operation was concluded even though the amount of parathyroid tissue removed seemed insufficient to account for the pronounced hypercalcemia.

Postoperatively the calcium levels remained elevated. On examination of the permanent sections it was clear that the right superior gland biopsy did not contain any parathyroid tissue, but was a remnant of a visceral arch. It was reasonably certain that the missing right superior gland was not in the neck. An extensive search did not disclose any sign of occult malignancy. With the aid of a selenomethionine parathyroid scan and a barium swallow (Fig. 1) the missing gland was presumptively localized to the posterior superior mediastinum.

Five months after the first exploration, in April 1970, a second operation was done through the previous cervical incision. A large 3.75 gm. gland was found in the posterior superior mediastinum and was excised with considerable difficulty, but without the need of splitting the sternum. The patient had an anomalous bifurcation of the common carotid occurring in the chest. The thyrocervical trunk arose from the external carotid artery. The vascular pedicle of the parathyroid tumor arose at the posterior inferior surface of the thyroid gland from the inferior thyroid artery. Since this was the fourth gland to be excised, a 30 mg. segment of parathyroid tumor was transplanted in the sternal head of the left sternocleidomastoid muscle and marked with a silver clip.

The tumor was composed of chief cells and contained many cysts lined with parathyroid cells. Postoperatively the serum calcium fell to 8.0 mg. and phosphorus rose to 4.4 mg. per 100 ml. The patient was started on 50,000 units of calciferol daily, which was gradually discontinued over a period of 3 months. One year later the serum calcium and phosphorus were normal.

Figure 1. Barium swallow in a patient with a 3.75 gm. aberrant parathyroid tumor, deep in the posterior mediastinum, displacing the esophagus to the right (of the reader). The metallic clip is on a branchial pouch remnant, misinterpreted as the superior parathyroid on frozen section during the first exploration. The large tumor was found and removed at a second exploration through the cervical excision.

In conclusion, the diagnosis of hyperparathyroidism depends on the accuracy of serum calcium and phosphorus determinations and a clinical evaluation to detect complications of this disease, such as calcium oxalate urinary crystalluria.

INDICATIONS FOR EXPLORATION

Once the diagnosis is on solid grounds, the patients who should be advised to have an exploration fall in two categories: patients with significant hypercalcemia (> 11 mg. per 100 ml.) or (> 2.5 mEq. Ca++ per liter), and patients with any complications of hyperparathyroidism. In regard to patients on chronic renal dialysis, parathyroidectomy is indicated when significant osteodystrophy, severe bone pain, or hypercalcemia occurs.

EXPLORATION

Since the rate of recurrence of hyperparathyroidism after the excision of the "adenoma" was reaching unacceptable proportions in published series, in 1965 we began to excise systematically all parathyroid tissue except for 50 to 60 mg. of well vascularized tissue in all our explorations. The reasons for this approach represented attempts:

1. to reduce the recurrence rate, and obviate the necessity for second and third explorations, with their inherent difficulties;

2. to take into consideration the increasing incidence of multiglandular disease;

3. to obviate the difficulties encountered in the differentiation of hyperplastic glands of normal weight from normal glands, on frozen section;

4. to give the surgeon irrefutable proof that he has visualized the parathyroid glands;

5. to provide the pathologist with invaluable material for the comparison of histopathological changes between the various glands.

Since a significantly elevated incidence of postoperative pancreatitis has been reported in these operations and since these patients are prone to peptic ulcers, preoperative antacid therapy and the avoidance of opiates in anesthetic premedication is advisable.

The technique is simple and standardized. A low cervical incision is used. The strap muscles are divided in the midline and retracted laterally; there is no need for the division of these muscles. The superior pole vessels and the middle thyroid vein are divided between ligatures; a suture-ligature is placed in the lateral portion of the thyroid lobe to facilitate retraction. All four glands are identified. The superior glands are usually located at the superior end of the recurrent nerves. The inferior glands vary considerably in location and can frequently be found within the substance of the thymus gland. Once all four glands are identified, the one which appears the most normal grossly is selected for preservation and biopsied in such a fashion that 50 to 60 mg. of tissue remains attached to the vascular pedicle, and marked with a metallic clip for future identification and localization, if required. If a gland is missing, the thyroid lobe is resected on the same side. The incision is closed without drains, except in patients on chronic dialysis. With a modicum of experience, there is no need for staining techniques (such as toluidine blue)[2] to identify the parathyroids, which are usually clearly brownish-tan, whereas the thyroid is reddish-brown. The hemostasis has to be meticulous. In 98 explorations we have had no occasion where a sternotomy was required to reach the aberrant glands in the superior mediastinum.

When the need arises, 50 to 75 mg. of parathyroid tissue can be transplanted easily in the sternal head of the sternocleidomastoid muscle, after cutting the parathyroid tissue into small slices and creating a small amount of bleeding in the small cavity made by spreading the muscle fibers.[8] Once the tissue is in the depth of the muscle, the fibers are reapproximated with a metallic clip, which serves as a marker for the future.

POSTOPERATIVE COURSE

The postoperative course is usually quite benign. Somewhere between 48 and 96 hours many patients will develop perioral numbness and mild tingling in their fingers, and will display a positive Chvostek sign. Usually these symptoms will be heralded by a negative urinary Sulkowitch. It is desirable to see the serum calcium drop to 7.5 or 8.0 mg. per

Table 2. *Number of Hyperparathyroid Glands Verified Histologically in 75 Patients Explored for Primary or Secondary (Renal) Hyperparathyroidism*

4 Glands examined	41
3 Glands examined	25
2 Glands examined	7
1 Gland examined	2

100 ml., as an indication that the parathyroidectomy was adequate. The symptoms of mild hypocalcemia are short-lived and the great majority of these patients are discharged within a few days after operation. If the symptoms of hypocalcemia become intolerable and the patient continues to require intravenous injections of calcium gluconate (the use of $CaCl_2$ is to be discouraged, because it is markedly irritant to tissues at the site of injection), then 50,000 units of calciferol may be administered orally daily and the patient discharged within a few days. Subsequently, as soon as the calcium and phosphorus have returned to the normal range, the calciferol can be tapered over a period of a few weeks.

The patients on chronic renal dialysis should be dialyzed the day prior to and the day after operation. Their wounds heal more slowly and often drain a clear transudate for a few days. They are able to tolerate their diet within 48 hours. It is advisable to treat them with an aluminum gel to buffer gastric acid and bind phosphates. The serum calcium may drop to very low levels in these patients. However, since they retain large quantities of calcium at the time of dialysis they seldom require calcium injections. The results in these patients are dramatic; the bone pain usually subsides in a short period of time.

RESULTS

The results are summarized in Tables 2, 3, and 4. The histological studies continue to indicate a high incidence of multiglandular disease, to such a degree that the excision of the single largest tumor alone subjects two thirds of patients to the possibility of continued disease or recurrence within a short period of time.

One of the failed explorations was described in Case 1. In a patient with total serum calciums in the 12 to 14 mg. per 100 ml. range, one should find at least 600 to 1,000 mg. of parathyroid tissue. Even though

Table 3. *Hyperparathyroidism (98 Explorations)*

Failed exploration (2)	2%
Permanent hypoparathyroidism (2)	2%
Recurrent nerve injury (1)	1%
Recurrence of disease (1)	1%
Mortality	0%

Table 4. *Hyperparathyroidism (98 Explorations)*

Primary hyperparathyroidism	71
Renal (secondary) hyperparathyroidism	13
Diagnostic problems	12
Second (intramural) explorations	2

we thought we had identified four glands by frozen section (on permanent section one of the four proved to be a branchial pouch remnant), the amount of tissue excised in that patient could not have accounted for the hypercalcemia, and therefore in retrospect we should have persisted and should have continued our exploration in search of a "fifth" gland. The second failure was in a patient with carcinoma of the breast, with a good possibility of non-documented metastases. The diagnosis may not have been correct since some months after failure to correct the hypercalcemia by exploration, there was a spontaneous fall of serum calcium to the normal range.

The two patients with permanent hypoparathyroidism are of interest. The first one had a simultaneous total thyroidectomy for a co-existing thyroid carcinoma. Apparently the vascular supply to the parathyroid remnant was inadequate. Such glands would have a better chance to remain viable and functional if transplanted into the sternocleidomastoid muscle. The second patient was 85 years old, when a 3½ parathyroidectomy was done. She has required three times the usual dose of calciferol to maintain eucalcemia. In an elderly patient one may have to leave a larger segment of parathyroid tissue, since there may be impairment of calcium absorption. The only recurrence is described in the following case.

Case 2

This 33 year old man with chronic renal disease had been on hemodialysis since 1968. During this period of time his serum calcium rose to the 8 to 11 mg. per 100 ml. range, while serum phosphates were in the 7 to 9 mg. per 100 ml. range; alkaline phosphatase was markedly elevated. Severe osteoporosis and subperiosteal resorption had developed in many bones. In addition there were large periarticular soft tissue calcifications in the shoulder and in the hands (Fig. 2). He had severe, incapacitating bone pain, especially in the legs. In June of 1970 a 3½ parathyroidectomy was done: two grams of parathyroid tissue were excised. Approximately 50 mg. of the left inferior gland were preserved. All four glands were hyperplastic, with "chief cell adenomatous" changes in one, superimposed to the hyperplasia.

Following operation, there was a marked improvement in symptomatology with resorption of the soft tissue calcifications and an increase in mineral content of the bones as measured by bone density.

However, within 1 year after 3½ parathyroidectomy, all the symptoms had recurred, the bone density had slipped back to a very low level, and the soft tissue calcifications had increased in dimension.

In June of 1971 the parathyroid remnant was excised in toto. It weighed approximately 120 mg; apparently it had doubled in size.

The results were much more dramatic than after the first operation, with marked reduction in the soft tissue calcifications and remission of his bone pain, to the point that for the first time in 4 years the patient returned to work.

Figure 2. Renal hyperparathyroidism. Severe calcium resorption is present throughout the bones of all the digits. The periarticular soft tissue metastatic calcifications are characteristic of renal hyperparathyroidism.

The current trend is to do a total parathyroidectomy in patients who are not candidates for transplantation and will remain on chronic dialysis, and 3½ parathyroidectomy in patients who will have renal transplants.

CONCLUSIONS

Hyperparathyroidism begins as a disease of at least two if not four parathyroid glands and remains so to a greater or lesser degree in the majority of patients by the time the disease is suspected or diagnosed with current methodology.

Near-total parathyroidectomy gives the best chance for a recurrence rate of zero in what is generally regarded as "primary hyperparathyroidism" and less than 10 per cent in patients on chronic renal dialysis. Serious consideration should be given to total parathyroidectomy in such patients, if they are not candidates for renal transplantation.

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Some Unusual Manifestations of Parathyroid Disease

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During the past 15 years, we have had the opportunity to study 51 patients with proven hyperparathyroidism at Chicago Wesley Memorial Hospital. Several of these patients had findings that are of unusual interest. It is these uncommon cases that form the basis of this report. In each of these cases, the highest serum calcium level and lowest inorganic phosphorus level are reported.

Symptoms Compatible with Brain Tumor

B.M., a 48 year old white woman, was admitted in March of 1970 with excruciating headaches, nausea, vomiting, lethargy, somnolescence, and hemiplegia. She was asphasic and had conjugate deviation of the eyes. Blood pressure was 190 mm. Hg systolic, 98 diastolic. Later she developed spinal rigidity and did not respond to painful stimuli. Her past history revealed chronic pyelonephritis with renal stones and severe constipation. The brain scan was compatible with the diagnosis of a brain tumor. Angiography was normal, although a possibility of tumor blushing was seen. Serum calcium was 17.8 mg., inorganic phosphorus 1.9 mg., and blood urea nitrogen was 47 mg. per 100 ml.

Mithramycin was given for 2 days, as the working diagnosis was that of a brain tumor. Inadvertently, the mithramycin helped reduce the serum calcium and the patient began to respond. The patient was given intravenous phosphates and steroids, and her general condition continued to improve. An urgent neck exploration was carried out, and a parathyroid adenoma was removed from behind the esophagus near the right lower pole of the thyroid. Twenty-four hours later, the serum calcium had dropped to 9 mg., and forty-eight hours after the operation it had dropped to 7.7 mg. per 100 ml. This later became stabilized at 9.5 mg. per 100 ml.

The patient aroused from the coma and little by little began to learn

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to speak. She was given psychological tests at serial intervals. Within a month she was able to converse at about a second grade level and writing was at about the same level. A year following her parathyroid surgery, the patient was able to speak normally and to read and write at an adult level. She is totally rehabilitated, carrying out her housework and leading a normal life.

Comment. This patient was thought to have a brain tumor. The high serum calcium level on the screening tests alerted the diagnostician to the correct diagnosis of hyperparathyroid crisis.[6,11]

Inability to Walk—A "Wall Walker"

R. A., a 46 year old black female school teacher, was admitted in 1968 because of diplopia, dizziness, and inability to stand or walk. For two months prior to admission she had had to place both hands on a wall in order to walk. Her speech was quite slurred. She was admitted with the diagnosis of probable brain tumor. Angiography, myelography, and encephalography were normal. Serum calcium was 15.0 mg. and serum inorganic phosphorus was 2.3 mg. per 100 ml. Her bone showed generalized deossification. The cortisone suppression tests failed to lower the serum calcium, and the calcium intake and output studies were indicative of hyperparathyroidism. Her neck was explored and a 1.5 gm. parathyroid adenoma was removed. The postoperative course was entirely uneventful. The patient gradually was able to resume walking, her diplopia disappeared, and her speech gradually returned to normal. It has been 4 years since her operation. She is employed full time as a school teacher and has no residual disability.

Comment. Her severe neuromuscular disabilities required rehabilitation, but the patient recovered completely. Both peripheral and central nervous systems are susceptible to disorders of calcium metabolism.

Multiple Endocrinopathies

M. G., a 34 year old white woman, was admitted in 1963 with renal disease due to polycystic kidneys and severe hypertension. Three months before admission this patient had been subjected to a bilateral adrenalectomy for Cushing's disease. At this admission the serum calcium was 15.4 mg., and the serum inorganic phosphorus was 1.7 mg. per 100 ml. The calcium intake and output studies were suggestive of hyperparathyroidism. The tubular reabsorption of phosphate was 60 per cent. The patient was subjected to a neck exploration and three parathyroid adenomas were removed, the largest weighing 2700 mg., the second 400 mg., and the third 140 mg. The patient responded well as far as calcium metabolism was concerned, but later developed pancreatic islet cell hyperplasia (insulinoma) and signs and symptoms of hyperpituitarism. Her renal disease was progressive and she died of renal failure in 1970.

Comment. This patient had polyendocrine disease involving the pituitary, adrenals, pancreas, and parathyroids. While other investigators have found multiple adenomas with regularity, we had only three known cases in our present series.[3,5] Admittedly, we rarely biopsied the "normal" glands when removing an obvious adenoma.

Parathyroid Adenoma in a Patient with Zollinger-Ellison Syndrome

C. M., a 53 year old white woman, was admitted with her third episode of severe peptic ulcer disease. Gastric function studies were compatible with Zollinger-Ellison syndrome. She had two brothers that had died of pancreatic carcinoma, and one brother died of a carcinoma of the stomach. Serum calcium was 12.6 mg. and inorganic phosphorus was 1.4 mg. per 100 ml. Hyperparathyroidism, as well as the Zollinger-Ellison syndrome, was diagnosed. A neck exploration was carried out in 1966 and two parathyroid adenomas were removed. Abdominal exploration revealed a tumor of the body of the pancreas. This proved to be a nonresectable pancreatic islet cell adenocarcinoma. Eighteen months later the patient died of disseminated pancreatic carcinoma.

Comment. This patient had a non-beta islet cell adenocarcinoma of the pancreas and two adenomas of the parathyroid glands. Four of the patient's 8 siblings had multiple endocrine disease. Patients with hyperparathyroidism have a higher incidence of Zollinger-Ellison syndrome than is seen in the general population.

Parathyroid Adenoma Associated with Follicular Carcinoma of the Thyroid

W. W., a 61 year old black man, was admitted in 1970 for repair of an inguinal hernia. He had no other symptoms. Physical examination revealed a nodule on the right side of his thyroid gland. The serum calcium was 13.0 mg. and the inorganic phosphorus 2.2 mg. per 100 ml. Cortisone failed to suppress the serum calcium levels. A thyroid scan revealed a functioning nodule on the right lobe of the thyroid. A neck exploration revealed a right lower parathyroid adenoma. The nodule on the right lobe of the thyroid was also excised and was reported as being a follicular adenoma. Two days later the thyroid tumor was reported as being a follicular adenocarcinoma. Ten days following the parathyroidectomy, a total thyroidectomy was carried out. His postoperative course was uneventful. A month later he finally had his right inguinal hernia repaired. He has been well for the ensuing 18 months.

Comment. Two of our 51 patients had follicular carcinoma of the thyroid diagnosed when exploring the neck for parathyroid adenoma. None of our patients had medullary thyroid carcinoma and none had pheochromocytoma.

Parathyroid Adenoma Associated with Ulcerative Colitis

D. C., a 49 year old woman, was admitted in 1968 with a severe exacerbation of known ulcerative colitis. She had a daily fever of 103° F., 7 stools a day, had lost 15 pounds, and had evidence of liver disease. On admission the serum calcium was 12.6 mg., and the inorganic phosphorus 1.4 mg. per 100 ml. Ten days after admission acute distention of the abdomen developed and an x-ray film was compatible with toxic megacolon. Within 4 hours she had signs and symptoms of peritonitis from colon perforation. An emergency abdominoperineal total colectomy was performed, and the abdomen drained. She had a stormy postoperative course. Two months later she still had intra-abdominal drainage and unhealed abdominal and perineal wounds. Her personality had deterio-

rated to that of a child. The serum calcium was 15.6 mg. and inorganic phosphorus 1.4 mg. per 100 ml. It was postulated that the inability to heal might be related to the hyperparathyroidism. Consequently, a neck exploration was performed and a 1300 mg. parathyroid adenoma was removed. Within days her general condition began to improve and within 3 weeks all drainage had ceased and the wounds were healed. Her personality reverted to that of a sound, cooperative patient. Because of the severe cachexia, a full year was required for the patient to regain her weight and strength. For the past 3 years she has been in excellent health and has resumed full work as a real estate saleslady while maintaining her own home.

Comment. To the best of our knowledge, this is the first case of hyperparathyroidism diagnosed in a patient with ulcerative colitis. Removing the parathyroid adenoma appeared to affect the healing of her wounds as well as produce a marked improvement in her personality.

Hyperparathyroidism in a Quadraplegic Adolescent

S. M., a 14 year old white boy, was in an automobile accident in September 1970 (4 months prior to this admission) and sustained a fracture of his cervical spine at the C6 level. He was made quadraplegic by this accident. Four days after the accident he had a spinal fusion. During the 4 months following this operation the patient regained some function of his right upper extremity. He was in the Chicago Rehabilitation Institute when an SMA 12 revealed a serum calcium of 15 mg. and an inorganic phosphorus of 2.0 mg. per 100 ml. He had had nausea, constipation, vomiting, had lost 35 lbs. in weight, and had developed kidney and bladder stones. He was severely depressed. He was admitted to Chicago Wesley Memorial Hospital, where tubular reabsorption of phosphate was 64 per cent and serum parathormone levels were 20 times higher than the normal range. Neck exploration revealed four enlarged parathyroid glands. Three and two-thirds of these glands were excised. Postoperatively, his serum calcium came down to 10.5 mg. per 100 ml. and has remained at this level for the past year. The symptoms of nausea, vomiting, constipation and renal stones all have disappeared. The depression is hard to evaluate, but seems to be improved. His weight has stabilized.

Comment. The mineral metabolism in adolescent quadraplegics is not well understood. In this patient, circulating serum parathormone levels were helpful in establishing diagnosis of hyperparathyroidism. Removing 3⅔ of the parathyroid glands relieved the symptoms often attributed to hyperparathyroidism.[5]

Bone Lesions in a Patient with Chronic Renal Disease and Breast Carcinoma

M. J., a 51 year old white woman, was admitted in 1971 with severe bone pain from lytic lesions in her left lower tibia and left upper humerus. This patient had had polycystic kidneys with chronic progressive renal failure for the past 20 years. A year and a half prior to this admission, she had had a radical mastectomy for carcinoma of the breast. Three of 20 axillary nodes were positive for carcinoma. Bone biopsy revealed osteitis fibrosis cystica. Circulating parathormone levels were markedly

elevated. The serum calcium was 11.0 mg. and serum inorganic phosphorus 8 mg. per 100 ml. A diagnosis of secondary hyperparathyroidism was made and subtotal parathyroidectomy was suggested, as the nephrologist felt that the hyperparathyroidism would contribute to her renal disease. A neck exploration revealed four enlarged parathyroids and 3½ were removed.There was no problem with hemorrhage during this procedure. Pathological examination of the parathyroid glands revealed hyperplasia. Postoperatively, she had marked relief from her bone pain. She is now on chronic renal dialysis.

Comment. In this patient, it would have been easy to have mistaken the bone lesions for metastatic breast carcinoma. Two bone types exist in azotemic bone disease. The first, osteomalacia or renal rickets, responds well to vitamin D. The second is associated with a normal or high serum calcium with osteoclastic formation of bone cysts. The latter is more rare, but will lead to soft tissue calcification and is a clear indication for parathyroid surgery.[2] Some surgeons have suggested total parathyroidectomy for these patients if they are to undergo chronic renal dialysis.

Carcinoma of Parathyroid

I. H., a 63 year old black woman, was admitted in 1969 with marked deossification of the bones. The high serum calcium was 17.5 mg. and the low serum inorganic phosphorus was 2.8 mg. per 100 ml. A neck exploration revealed three normal-appearing parathyroid glands and an enlarged left superior gland that was 4 cm. in its greatest length and was quite firm on section. The frozen section diagnosis was parathyroid adenoma. Two days later the paraffin sections were reported as a parathyroid carcinoma. The patient was re-operated upon and a left radical neck dissection was carried out. Serial sections of the lymph nodes failed to demonstrate tumor metastasis. Postoperatively, the serum calcium dropped to 7.7 mg., but stabilized later at 9.5 to 10 mg. per 100 ml. During the postoperative period, a bleeding peptic ulcer developed and was treated by medical management.

A year later the patient was admitted with a mass in the suprascapular region of her back. A biopsy was taken and the tumor was diagnosed as fibrosarcoma. In spite of intensive irradiation therapy, the patient died in 18 months from disseminated fibrosarcoma. At the autopsy there was no evidence of recurrent or metastatic parathyroid tumor.

Comment. This is the only patient in our series that had a malignancy of the parathyroid gland. Over 90 per cent of parathyroid carcinomas are functioning and only about 15 per cent survive 5 years.[8]

Postoperative Death—Possible Hypomagnesemia

L. R., a 71 year old white woman was admitted to another hospital 17 months before being admitted to Wesley. At that time her symptoms were fatigue, constipation, and polyuria. The past history revealed that she had two operations on her thyroid for a toxic nodular goiter. She was told that she had hyperparathyroidism, but she refused an operation. Five months later she noticed painful swelling of her right cheek and of the left ankle. These turned out to be bone cysts, compatible with osteitis fibrosis cystica. At the current admission the laboratory studies were: serum alkaline

phosphatase 2100, blood urea nitrogen 23, serum calcium 16.8 mg., and the inorganic phosphorus 2.5 mg. per 100 ml. A bone survey showed osteoclastic lesions in the skull, clavicles, radius, ulna, and pubic bones, as well as the zygomatic arch and lower tibia. A serum magnesium level was 1.0 mg. per 100 ml., the normal being 1.8 to 3. She was given magnesium sulfate prior to operation. A parathyroid adenoma was removed from the left upper portion of the remaining thyroid tissue. Twenty-four hours following this operation, her serum calcium was reported as 9.1 mg. per 100 ml. She had a fever of 102 F. On the evening of the first postoperative day, the patient had an episode of wild uncontrollable behavior. She complained of pain all over her body. At 10 P.M. of the first postoperative day, the patient became apneic and the monitor showed asystole. Serum calcium at this time was 8.5 mg. per 100 ml. She was resuscitated and was given calcium gluconate, albumin, and aramine. Her pupils became enlarged and fixed and she became anuric. She died 48 hours after the parathyroid tumor was removed. Serum magnesium levels were not taken during this period. Post mortem examination was refused.

Comment. This is the only postoperative death in this series of 51 patients. While this patient may have died of pulmonary emboli or other causes, the possibility of hypomagnesemia exists. It has been reported that during the first 24 to 48 hours following parathyroidectomy, patients with severe hypercalcemia and marked cystic bone lesions may lose magnesium as well as calcium into the bone cysts. Hypomagnesemia can lead to cardiac irregularities and death. Central nervous system symptoms are often prominent, especially severe agitation.[1, 7, 10]

Adenoma in an Unusual Location

L.M., a 39 year old black man, was admitted in 1961 with recurrent renal stones, severe constipation, hypercalcemia, and hypophosphatemia. A prednisone suppression test failed to suppress the high serum calcium. Serum calcium intake and output studies were compatible with hyperparathyroidism. He had a neck exploration and two normal parathyroids were biopsied. Three months later the patient was re-explored and a third normal parathyroid gland was biopsied along with a large portion of the thyroid gland. The hyperparathyroidism persisted. At the time of the third admission in 1962, he had a serum calcium of 15.9 mg. and a serum inorganic phosphorus of 1.8 mg. per 100 ml. He still had severe constipation and renal stones. The one gland that had not been found was the right superior gland. At the third operation blood vessels leading behind the pharynx were noted. The total area was mobilized so that the index fingers could meet behind the pharynx. A parathyroid adenoma weighing 1100 mg. was found deep to the pharyngeal constrictor muscles and was removed. The patient had temporary hypoparathyroidism, a condition which corrected itself without medication. Since then the patient has had normal serum calcium levels. He takes two grains of thyroid a day, but is otherwise healthy. His last renal stone was found 8 years ago.

Comment. It is not uncommon for a parathyroid gland to migrate behind the esophagus; 6 of our 51 patients had parathyroid adenomas in this area. However, this is the only case we can document that had an adenoma behind the *pharynx*. Parathyroid adenomas often carry their

blood supply with them and in this case the blood vessels led to the discovery of the adenoma.

Another patient, A. S., a 73 year old white woman was admitted in 1961 with nephrolithiasis, nephrocalcinosis, hypertension, and symptoms of a recent cerebral hemorrhage. She had had progressive memory loss, headaches, and confusion for a month prior to admission. She died the day after she was admitted and the autopsy revealed a massive cerebral hemorrhage. In addition, a 2.5 cm. parathyroid adenoma was found in the lower anterior mediastinum just above the diaphragm. To the best of our knowledge, a tumor in this position has not been described in the surgical literature. This case was not included in this series as there was no laboratory proof that this was a functioning tumor. However, it can be postulated that the parathyroid tumor could have caused the renal disease and the hypertension which led to her death.

Substernal Adenoma

D. G., a 66 year old white woman, was admitted in 1961 with bone cysts and nephrolithiasis. The serum calcium was 14.5 mg. and the inorganic phosphorus 2.0 mg. per 100 ml. The tubular reabsorption of phosphate was 79 per cent. This patient had renal disease and appeared much older than her stated age. A neck exploration was carried out and three normal parathyroid glands were identified. The left inferior gland could not be found. The superior mediastinum could not be satisfactorily explored through her collar incision. The left lobe of the thyroid was excised, hoping to remove the left inferior parathyroid. While her immediate postoperative course was satisfactory, her situation did not improve. The serum calcium never dropped below 12 mg. per 100 ml. Permission to explore the superior mediastinum could not be obtained. The patient lived a year and a half and died of progressive renal failure. An autopsy revealed a parathyroid adenoma on the arch of the aorta which weighed 34 gm.

Comment. Most substernal parathyroid adenomas can be removed through the usual collar incisions.[9] Indeed, 3 of our 51 patients had parathyroid adenomas removed from the superior mediastinum through these incisions. In this particular case it might have been difficult to remove this large tumor through a neck incision, but it would certainly have been easy to remove it with a sternal-splitting incision.

DISCUSSION

The purpose of this paper is to describe some of the uncommon findings in patients with parathyroid disease. The series of cases from which they derived are as follows:

Patient population. The patients were from 14 to 80 years in age, the average being 50 years. There were 42 white and 9 black patients. There were 33 women and 18 men. The duration of symptoms could not be estimated with accuracy. Case reports of two of these patients (R. A. and D. C.) will appear in greater detail at a later time.

Glands removed. Twenty-five adenomas were weighed. They ranged from 200 mg. to 34 gm. The average weight was 3.5 gm., but most of the adenomas weighed between 1 and 2 gm.

Table 1. *Types of Symptoms Thought to Be Related to Hyperparathyroidism in 51 Patients*

Renal (27 patients)	Stones	22
	Pyelonephritis	2
	Nephrocalcinosis	2
	Dysuria	2
Bone (20 patients)	Cysts	6
	Deossification	11
	Bone pain	3
Hypertension (16 patients)		
Gastrointestinal (14 patients)	Abdominal pain	4
	Peptic ulcers	3
	Nausea and vomiting	3
	Constipation	3
	Pancreatitis	1
Neuromuscular (11 patients)		
Multiple Endocrine (6 patients)	Pancreatic adenomas	1
	Malignant Zollinger-Ellison	1
	Follicular carcinoma of thyroid	2
	Cushing's, islet cell, hyperpituitarism	1
	Hyperthyroid	1
Emotional distress (6 patients)		
Severe Depression and Confusion (3 patients)		
Headache (3 patients)		
Cataracts (1 patient)		

Symptoms. The symptoms referable to hyperparathyroidism are shown in Table 1. The frequency of symptoms is not unlike most American series, except that hypertension and neuromuscular disabilities were found more frequently in this series. Peptic ulcers appear in about the same proportion as are found in the general population.

Laboratory diagnosis. In our series, the serial serum calcium and inorganic phosphorus levels most accurately reflected the actual pathological findings. The cortisone suppression test was quite useful. The tubular reabsorption of phosphate was worthwhile in most cases. The calcium balance studies were difficult, but usually were accurate. Serum parathormone levels were closely related to the pathological findings. Parathyroid scans and C 47 absorption tests have been discontinued at this time. At this time our routine examinations include serial serum calcium and phosphorus determinations, a cortisone suppression test, and the tubular reabsorption of phosphate. In difficult cases, serum parathormone levels are obtained (see Table 2)

Table 2. *Diagnostic Tests in 51 Patients with Proven Hyperparathyroidism*

Highest serum calcium	11.3 to 18.8 (average, 13.1 mg. per 100 ml.)	
Lowest inorganic phosphorus	1.0 to 2.8 (average, 1.7 mg. per 100 ml.)	
Tubular reabsorption of phosphate (correlation to hyperparathyroidism)		
	correct	12/22
	incorrect	6/22
	equivocal	4/22
Selenium scans	6 cases (not helpful in 5)	
Cortisone suppression test	helpful in 18 of 20	
Calcium-47 absorption	3 patients (not helpful)	
Serum parathormone	6 patients (helpful in each case)	
Calcium intake and output	correct	14/19
	not diagnostic	5/19

The type of hyperparathyroidism is known in Table 3. We have had fewer cases of hyperplasia and fewer multiple adenomas than in other series.[5] This may well have been due to our reluctance to operate on equivocal cases, or our hesitance to biopsy what appear to be normal glands. We have found that a functioning parathyroid adenoma is rarely associated with atrophy of the remaining glands. In our experience the remaining glands usually appeared grossly normal. We agree that any questionably enlarged gland should be biopsied. Because of the frequency of multiple gland involvement, some surgeons suggest that 3½ glands be removed in all cases of hyperparathyroidism (see Dr. Paloyan's article in this issue).

Table 3. *Type of Hyperparathyroidism in 51 Patients*

	NO. OF PATIENTS
Solitary adenomas	43 (84%)
Multiple adenomas	3 (6%)
Hyperplasia	2 (4%)
Not accurately recorded	2 (4%)
Carcinoma	1 (2%)

The location of solitary adenomas is recorded in Table 4. This is similar to the findings in most series.

Table 4. *Location of Solitary Adenomas*

	NO. OF PATIENTS
Right superior	6 (14%)
Left superior	6 (14%)
Right inferior	14 (32%)
Left inferior	17 (40%)

Table 5. *Unusual Location of Solitary Adenomas in 11 Patients*

Behind esophagus (6 patients)	Right superior	2
	Right inferior	3
	Left inferior	1
Behind pharynx (1 patient)	Right superior	
Anterior mediastinum (3 patients) (below notch)	Right inferior	1
	Left inferior	2
Arch of aorta (1 patient)	Left inferior	

Some of the unusual locations of parathyroid adenomas are shown in Table 5.

Recurrence. To the best of our knowledge, none of the patients in this series has had recurrence of hyperparathyroidism. Six have been lost to follow-up. Recurrences have been found in some series in the area of 30 per cent.[3, 4]

SUMMARY

Hyperparathyroidism can masquerade as many diseases, can be associated with many diseases, and can present technical difficulties in the surgical removal of the diseased glands. Twelve cases are presented that reflect some of the interesting problems in each of these areas.

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Aneurysms of the Common Carotid Artery

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Every physician will encounter symptomatic patients with pulsating and occlusive vascular diseases of the neck vessels. Because the surgical treatment of these is gratifying and beneficial, a detailed consideration of one entity, aneurysms of the common carotid artery, is useful.

Carotid aneurysms constitute less than 10 per cent of all peripheral aneurysms. Among 2300 operations performed at Baylor University for peripheral aneurysms, only 7 involved the carotid arteries.[4] In 221 peripheral aneurysms (traumatic) incurred during World War II there were only 13 involving the common carotid artery.[29] Carotid aneurysms are generally fusiform, although saccular aneurysms may occur.[1, 2, 19, 22, 30]

Aneurysms of the common carotid artery are rare. However, aneurysms of the common carotid are twice as frequent as those of either the internal or external carotid arteries.

At present trauma and arteriosclerosis are the most common causes, although in the past syphilis and pharyngeal infections predominated. Tuberculosis and mycosis are uncommon causes.[20, 31] Cystic medial necrosis may involve the carotid arteries (dilatation and aneurysm formation) but infrequently in contrast to the aortic manifestations. It is of interest that the media of peripheral vessels are usually spared in Marfan's syndrome.[3, 9, 15, 17, 24, 33]

Infections and dissections of the common carotid artery after percutaneous angiography, owing to the subintimal injection of contrast medium, etc., have been noted and are unfortunate complications of these procedures.[6] Most recently the development of aneurysms following surgical reconstruction or endarterectomy of the carotid artery for occlusive disease or injuries, with either prosthetic or venous patches, has been reported.[10, 11, 25, 28, 32]

Congenital aneurysms have been described, but are exceedingly rare.[26]

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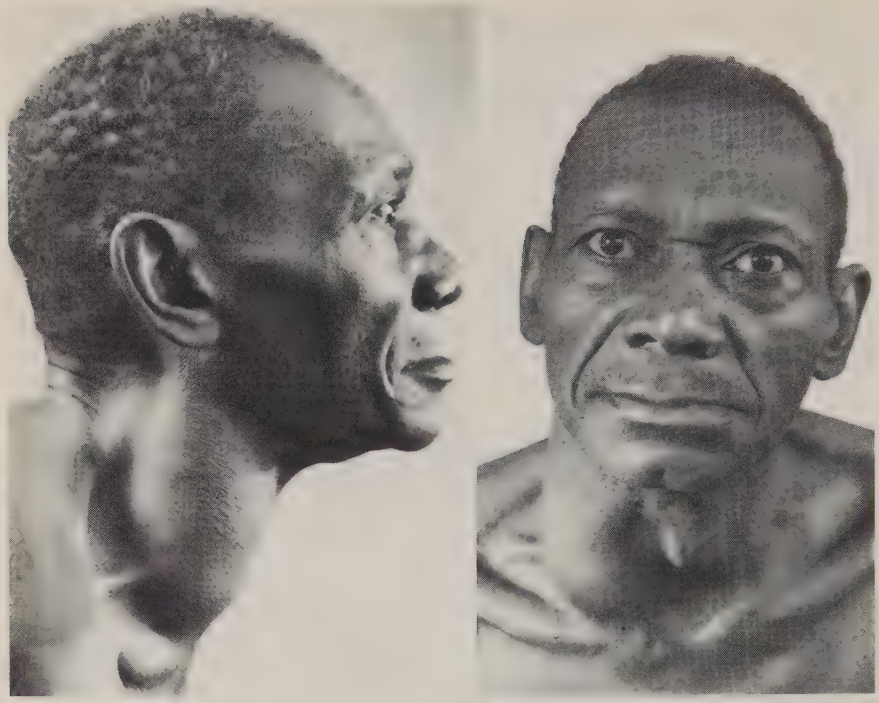

Figure 1. Appearance of a common carotid artery aneurysm in a 70 year old man who had noted a gradually enlarging mass in the right side of his neck for 6 months. Serology was positive. The mass decreased slightly in size with the administration of penicillin.

DIAGNOSIS

Common carotid artery aneurysms usually present as asymptomatic, pulsatile lateral cervical masses (Fig. 1) with a systolic bruit. With continued growth, extrinsic pressure symptoms and pain develop. An intrinsic pharyngeal component may be present, and many tragic results have been encountered by the confusion of a carotid aneurysm with a peritonsillar abscess. Embolic phenomena from the turbulent flow within the aneurysm and transient cerebral ischemia with subsequent neurologic deficit often occur.[13, 21, 34]

To be considered in the differential diagnosis of a pulsating cervical mass are thyroid tumors, branchial cleft cysts, lymphadenopathy, chemodectomas, and, most importantly, kinking of the carotid or innominate arteries.[5, 12, 18] It is stated that a kinked or buckled carotid or innominate artery will decrease in size and pulsation or disappear completely if the patient inspires and holds his breath—in contrast to an aneurysm of the carotid artery which continues to pulsate and remains constant in size.[7] Kinking of the carotid artery generally occurs in middle-aged women with hypertension and is uncommon in men. It should be considered as an occlusive disease of these vessels and may cause cerebral symptoms

by embolic phenomena or transient ischemic episodes.[27] Many instances of straightening the artery by resection and reanastomosis have been reported with relief of symptoms.

Traumatic aneurysms develop slowly and often their only clue is a small, scarcely noticeable scar. Arteriovenous fistulas are easily distinguished by the distended veins and characteristic bruit.

Once an aneurysm is suspected, angiography done in a retrograde fashion is the best and most definitive diagnostic study (Fig. 2). The exact extent of the aneurysm may or may not be visualized according to the amount of mural thrombus within the aneurysm (Fig. 3). If possible, the cerebral vessels should be outlined to determine if the internal carotid artery is patent and if other vascular anomalies are present. Either biplane angiography or views in several positions will delineate kinking of the carotid artery (Fig. 4).

Once diagnosed the recommended treatment for carotid aneurysms is excision. If a fusiform aneurysm is present, replacement by a prosthesis should be done. A saccular aneurysm can be transected through its stalk with or without use of a patch. Restoration of internal carotid flow is a mandate for carotid aneurysm surgery and ligation should be done only in unusual and selected instances (Fig. 5).

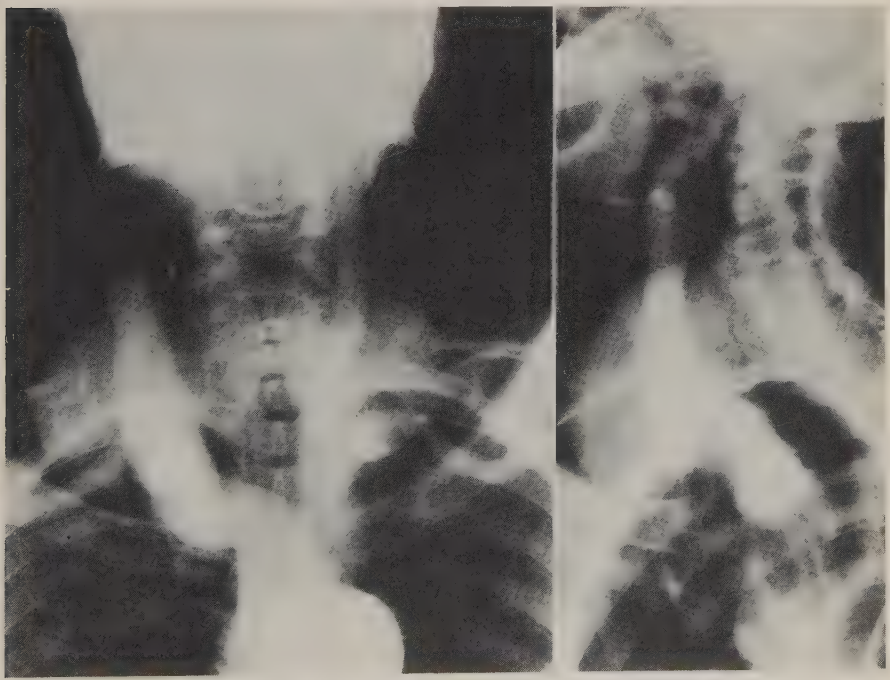

Figure 2. Retrograde aortogram via percutaneous femoral artery puncture. Note the generalized dilatation of the major branches of the aorta with aneurysmal dilatation of the right common carotid artery (same patient as in Figure 1).

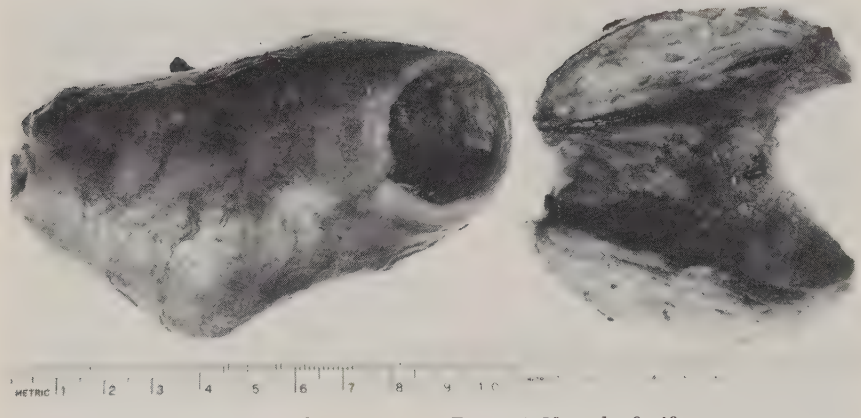

Figure 3. Gross specimen from patient in Figure 1. Note the fusiform appearance and the mural thrombus.

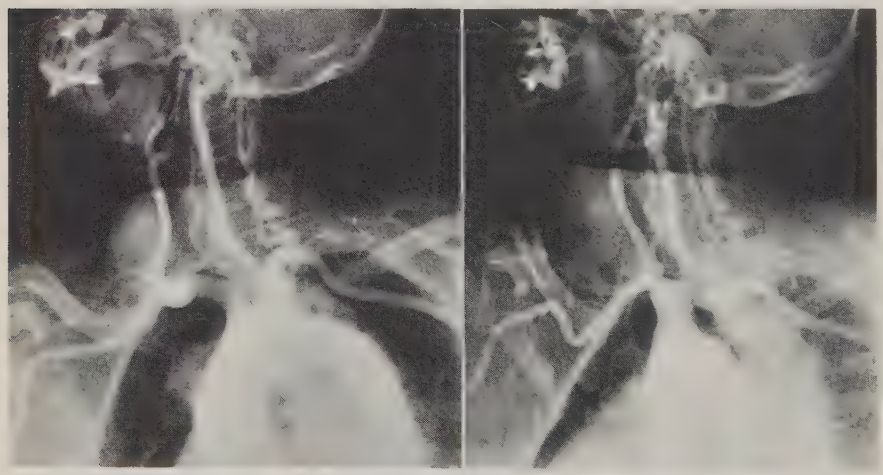

Figure 4. Angiograms of a 67 year old hypertensive woman thought to have a carotid aneurysm on physical examination. By comparing views in two positions, the kinking and tortuosity of the innominate and common carotid arteries are apparent. This presented clinically as a supraclavicular pulsatile mass. Operation is indicated if cerebral ischemic episodes occur.

Figure 5. This 59 year old man had an occluding clamp placed on his right common carotid artery for treatment of a right middle cerebral artery aneurysm. Two weeks later he presented febrile with the history of an enlarging pulsatile mass in the right neck. The angiogram shows the infected hematoma/aneurysm. In the absence of distal internal carotid flow and the presence of infection (Staphylococcus) the common carotid artery was sutured proximally. His postoperative course, now 3 years, has been uneventful.

OPERATIVE TECHNIQUE FOR COMMON CAROTID ANEURYSM

The patient under endotracheally administered general anesthesia is placed in a supine position with the neck hyperextended and the head deviated to the uninvolved side. Local anesthesia or regional cervical block can be used if the aneurysm is small and one can be certain that an extensive procedure will not be required. The most satisfactory incision is oblique and parallel to the anterior border of the sternocleidomastoid muscle. Dissection proximal to the aneurysm should be done first. If the aneurysm is localized within the neck, control of bleeding can be obtained by isolating and passing a Dacron tape around the common carotid artery. If the aneurysm is large (Fig. 6) the incision is extended as a midline sternotomy so that either the innominate or the left carotid artery can be isolated at its origin from the aortic arch. It is highly desirable to place a Dacron tape about the vessel for ease of manipulation and access.

After securing proximal control the aneurysm is delineated distally.

Appropriate care should be taken to preserve the vagus and phrenic nerves and the internal jugular vein, although the latter can be sacrificed for additional exposure. The internal and external carotid arteries are dissected sufficiently and Dacron tapes passed about them.

Once the extent of the aneurysm has been defined, it can be determined whether or not a clamp can be placed across the common carotid artery distally. If this is possible then cerebral blood flow can be maintained retrograde and the use of an internal carotid to common carotid shunt (either internal or external) is not necessary. If distal clamping of

Figure 6. Operative field during excision of common carotid aneurysm (patient shown in Figure 1). The cervical incision was extended as a midline sternotomy. Note tapes around the innominate artery proximally and the common carotid artery distally.

the common carotid artery cannot be done, then using a plastic shunt from the common carotid, innominate, or the arch of the aorta to the internal carotid distally is recommended. According to the anatomy of the aneurysm it may be best to insert a by-pass graft (Dacron) and then excise the aneurysm.

In general, end-to-end continuity is restored, although with large aneurysms end-to-side bypass restoration may be more feasible. Internal shunts are generally used when end-to-side reconstruction is done, and external shunts for end-to-end anastomoses.[8, 14, 16, 23]

The internal carotid artery should not be clamped for more than a few minutes because of the individual variation in tolerance to this occlusion. In one instance a neurologic deficit, although temporary, was experienced after only 4 minutes occlusion for repair of a common carotid injury in a young soldier (PGC). The vagaries of collateral circulation and the impositions of hypothermia and hypercarbia are such that use of a shunt, except when the external carotid can supply the internal carotid, is the method of choice. This allows careful reconstruction of the carotid without risk of neurologic damage.

Once distal and proximal occluding clamps have been placed, the aneurysm is resected, in contrast to the method generally used for abdominal aneurysms. Continuity is restored by insertion of a prosthetic preclotted graft (knitted Dacron) (Fig. 7). A continuous suture, or perhaps

Figure 7. Operative field after excision of aneurysm and restoration of internal carotid artery flow by insertion of a Dacron graft. The common carotid artery could be clamped distally so that no (external or internal) shunt was necessary. Patient is living and well 18 months after operation.

two, of 000 or 0000 suture material, preferably synthetic, is used. After the anastomosis has been almost completed distally, air is expressed from within the graft by releasing the proximal clamp. An autogenous saphenous or jugular vein graft can be used if of adequate diameter, but generally diseases producing aneurysmal dilatation also enlarge the common carotid to a size much greater than that of the saphenous vein. Venous patches can be obtained from the jugular vein.

The saccular aneurysm is clamped at its base. According to the anatomy, there may be a stalk sufficient to suture or it may be desirable to insert a venous or prosthetic arterial patch (gusset) to maintain carotid artery adequacy.

Excision of the aneurysm and restoration of cerebral flow is the standard form of therapy. The operation provides minimal trauma to the patient, and can be used in patients with associated cardiac, pulmonary, and renal disease with virtually no mortality or morbidity. Reports of long-term results, although not numerous, are uniformly gratifying.[4, 19, 22, 29]

A special problem in carotid artery surgery is presented by the dissections that occur as a result of the subintimal injection of dye during carotid arteriography. If there is any indication of progression, these should be resected since they may continue to dissect (Fig. 8).

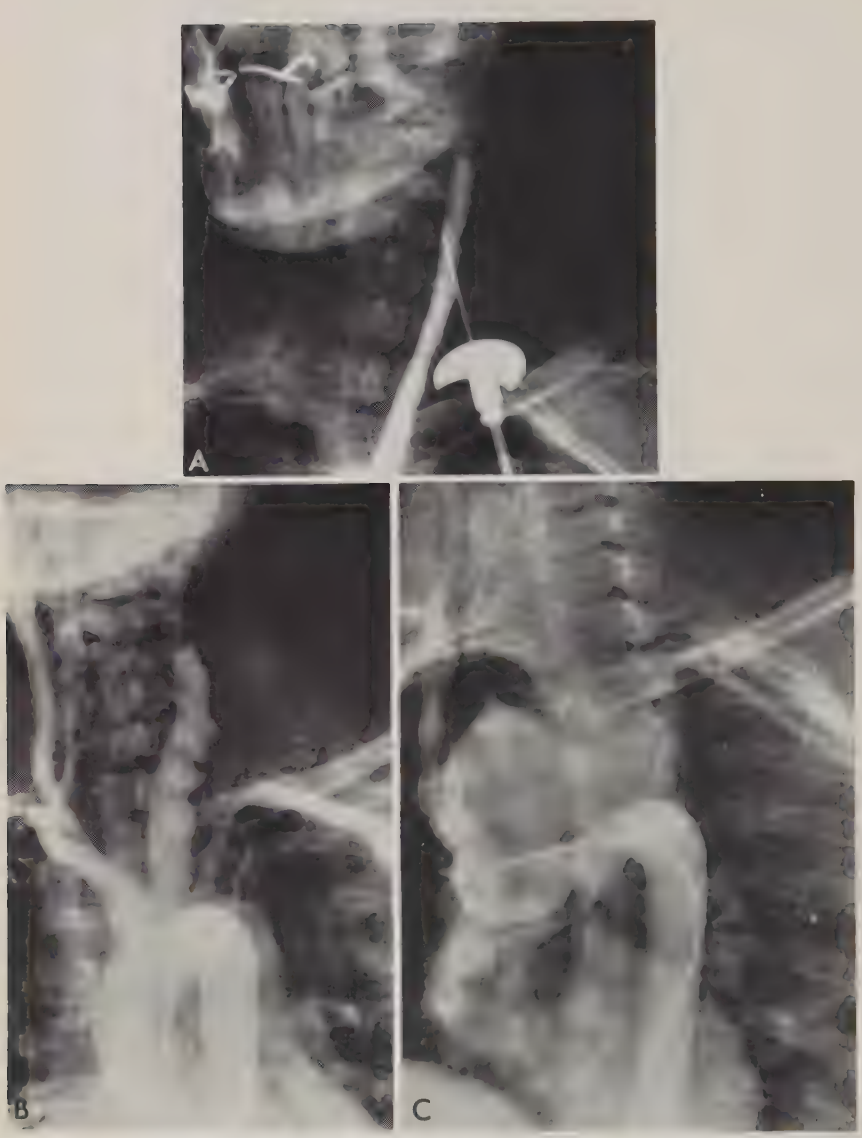

Figure 8. *A*, A 50 year old female with suspected subdural hematoma. Percutaneous carotid arteriogram caused subintimal dissection of the left common carotid artery that subsequently became infected (Staphylococcus aureus). *B*, Same patient, 10 days later. Retrograde aortography shows further dissection and dilatation of common carotid artery. Treatment consisted of antibiotic therapy. *C*, 17 days after original study. Infection and aneurysm (mycotic) progressed to involve the arch. Attempted repair at this time was unsuccessful.

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Surgical Correction of Carotid and Vertebral Artery Stenosis

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During the nineteenth century the relationship between occlusive vascular disease and stroke evolved. In 1828 Ambercrombie[1] observed that cerebral infarction was a disease of the aged and was analogous to gangrene in a limb when its blood supply was compromised. Even the early English literature made reference to the ravages of cerebral vascular insufficiency as shown in a poem by Swift (reproduced in the book *Stroke* by Irving Page, 1961):

> Poor gentleman, he droops apace,
> You plainly see it in his face,
> That old vertigo in his head
> Will never leave him til he's dead.
> Besides, his memory decays,
> He recollects not what he says
> For poetry he's past his prime,
> He takes an hour to find a rime;
> His fire is out, his wit decay'd
> His fancy sunk, his muse a Jade.
> I'd have him throw away his pen,
> But there's no talking to some men.

During the next century intracranial rather than extracranial factors were sought as the probable cause, until 1914 when Hunt[19] brought attention to the relationship of carotid artery obstruction to attacks of hemiplegia. It was not until 1928[32] with the development of cerebral angiography that this relationship could be documented in the living patient. It has only been in the past two decades that relief of transient ischemic attacks and prevention of cerebral infarction has been realized with the advent of revascularization of the extracranial cerebral vessels.[11]

Strokes are the third leading cause of death in the United States, resulting in the death of over 200,000 people per year.[2] Added to this is

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the 50 per cent morbidity of the people who survive the acute episode, of which only about 20 per cent return to full time work.[28] It has been estimated that in 1958 two million living Americans, half below the age of 60, were afflicted with strokes.[12]

Many studies[7,29,31] have shown that about 75 per cent of patients with completed strokes have had previous symptoms of transient ischemic attacks (TIA) and that about 40 per cent of these patients have had a significant obstruction of greater than 50 per cent of the internal carotid artery which was amenable to surgical repair.[8,17,30] One can only speculate as to the reduction of strokes had the lesion been repaired, but it does point out that most stroke patients have had premonitory symptoms and many of these patients have had correctable lesions.

The highest incidence of cerebrovascular insufficiency occurs between the fifth and seventh decades with men outnumbering women 2:1. The most common cause of extracranial cerebrovascular obstruction is atherosclerosis; and the most common site of involvement is the carotid bifurcation, which is the sole site in 30 per cent of the cases. If one takes into account that 65 to 75 per cent of patients have multiple lesions, then 75 per cent of surgically accessible lesions are found at the carotid bifurcation.[21,26,41] The second most common site of involvement with atherosclerosis is the proximal vertebral artery which is the sole site of obstruction in 10 per cent of patients.[21,26] The third most common site of vascular obstruction is the origin of the great vessels at the aortic arch. This is the sole site of involvement in 7 per cent of the patients with surgically accessible lesions.[26] There are two types: those found in the older population secondary to atherosclerosis and usually associated with lesions involving other cerebral vessels, and those lesions found in the younger population, usually women, caused by an inflammatory arteritis.

TYPES OF CEREBRAL DYSFUNCTION

Brachiocephalic cerebral vascular insufficiency, whether it be caused by atherosclerosis, fibromuscular hyperplasia, kinking of a tortuous artery, arteritis, embolus, or dissecting aneurysm of the aorta, can present with varying degrees of neurologic deficit. In general the types of dysfunction can be classified as follows:

1. *Transient ischemic attack (TIA)*, the mildest form of deficit, is a sudden cerebral episode which results in a distinct neurologic deficit, such as paresis, paresthesias, dizziness, or vertigo, usually lasting minutes to several hours but completely subsiding within 24 hours.

2. *Progressing stroke* is a persistent progressive neurologic deficit which becomes steadily worse over a several day period.

3. *Completed stroke* is a fixed static neurologic deficit.

Symptoms depend on the nature of the lesion, the location, extent of obstruction, and the collateral circulation. Because of these variables no clinical complex of symptoms is absolutely diagnostic of the site of the lesion.

The syndrome of *carotid artery insufficiency* classically presents

with weakness or numbness related to one side of the body (64 per cent of patients), speech difficulties if the dominant hemisphere is involved (21 per cent), dizziness (23 per cent), impaired vision on the side of the lesion (38 per cent). The visual disturbance, amaurosis fugax (fleeting blindness), is described by the patient as a shade or curtain obscuring his vision or transient loss of vision in the involved eye. Loss of consciousness is uncommon, occurring in less than 15 per cent of patients.

Vertebrobasilar insufficiency, which can be caused by either carotid or vertebral artery disease, may have symptoms similar to carotid artery insufficiency although the weakness and paresthesias are more likely to involve both sides of the body rather than one, have bilateral defects in the visual fields or bilateral blurring, and may exhibit diplopia, vertigo, or nystagmus.

Aortic arch obstructive disease does not usually present with focal neurologic loss as described above. With the proximal arterial obstruction and the rich distal collateral bed, a general reduction in cerebral blood flow occurs which is evenly distributed over the entire cerebral dysfunction, resulting in convulsions and syncopal episodes as well as upper extremity symptoms.

CLINICAL SIGNS

Physical examination is most helpful to accurately diagnose the aortic arch syndrome and the occasional vascular steal associated with it. When examining a patient with cerebrovascular insufficiency one must keep in mind the two mechanisms which can cause symptoms with obstruction of the great vessels, diminished forward flow and reversal of flow. Obstruction of the common carotid arteries with diminished forward flow and its attendant weak distal pulses can be readily diagnosed; however, the opportunity for reversal of flow from the internal to the external carotid artery exists. With obstruction of the innominate artery, forward flow is decreased and the right carotid and subclavian arteries will again have diminished pulsations; however, the potential for the reversal of flow, blood flowing proximally in the right carotid and vertebral artery, exists. Clinically this steal can be documented when one finds a weak right common carotid pulse and a reduction in the right brachial blood pressure which is accentuated by compression of the right carotid artery. Obstruction of either subclavian artery at its orgin can easily be diagnosed by noting the decreased brachial blood pressure. When reversal of blood flow through the vertebrobasilar system into the more distal subclavian artery is present, the diminution of the brachial pressure on the side of the lesion is accentuated by compression of either carotid artery while the blood pressure in the opposite extremity remains constant. This decrease in blood pressure is picked up clinically by noting the disappearance of the radial pulse with compression of the carotid artery. Bruits heard over the origins of these vessels will help substantiate the diagnosis when the vessel is partially occluded but will not be present when the vessel is completely occluded.

Cerebrovascular insufficiency from obstructive disease at the carotid bifurcation is suggested by palpation of a firm bifurcation and hearing a bruit. A thrill may or may not be present. A systolic bruit over this area is due to stenosis of the internal carotid in 90 per cent of patients; in 10 per cent the bruit is due to stenosis of the external carotid artery. The loudness of a bruit is related to the degree of stenosis and will increase in intensity as the intraluminal obstruction approaches 85 per cent, which corresponds to a vessel diameter of 1 to 1.5 mm.[41] As the stenotic lesion approaches the diameter of the artery, the bruit may become continuous depending on the collateral circulation (i.e., a significant gradient is present in both systole and diastole); or the bruit may disappear completely. Absence of a bruit occurs in about 20 per cent of patients with significant obstruction of the internal carotid artery, although half of these patients have complete occlusion.[10,41,45] Rotation of the neck with the production of a bruit and transient ischemic attack is suggestive of a kinked or tortuous artery.

TIA from vertebrobasilar arterial insufficiency can be due to either acute or chronic obstruction of the vertebral artery. The chronic form caused by atherosclerosis of the extra vertebral portion of the vertebral artery is commonly associated with stenosis of the internal carotid. Isolated vertebral artery stenosis is uncommon and accounts for 10 per cent of the extracranial occlusive vascular disease, and may be associated with a bruit over the base of the neck or the supraclavicular area.

Acute occlusion of the vertebral artery is due to external vertical or torsion stresses when the neck is turned and usually occurs at that part of the artery proximal to the vertebral canal. These patients are younger than those with atherosclerosis, with an average age of 40 years, and both sexes are equally afflicted. It has been estimated that 20 per cent of patients with vertebral artery insufficiency and without carotid artery disease have this syndrome.[36] Several mechanisms have been proposed: cervical arthritis[42] or spondylosis,[39] abnormal origin of the vertebral artery,[35] and the most common mechanisms, contraction of the longus coli, scalene anticus muscles or fascial bands of those muscles at the level of the vertebral foramen of the sixth cervical vertebra.[16,20] Symptoms tend to be more severe in these patients than those with chronic obstruction because the acuteness of the obstruction does not allow an adequate collateral circulation; added to this is the abnormal collateral circulation found in 95 per cent of these patients.[20] The opposite vertebral artery may have a small diameter, poor or nondemonstrable communication with the basilar artery, intrinsic disease of the opposite vertebral artery, carotid artery disease, incomplete circle of Willis, which is complete in only 50 per cent of the general population, or anomaly of the posterior communicating arteries which is present in 23 per cent of the general population.[4] Symptoms are precipitated when the collateral circulation cannot compensate for the acute decrease in cerebral blood flow. Treatment can be afforded by relief of obstruction of the carotid artery when present or release of the obstructing band.

PATHOPHYSIOLOGY

As has been previously mentioned, TIA is a focal or occasionally generalized neurologic dysfunction lasting several minutes to hours at the end of which time no neurologic deficit persists. Pathophysiologically this is a temporary reversible deficit of cerebral blood flow which produces symptoms when the blood supply fails to meet the metabolic demands of the tissue.

The severity of symptoms, i.e., cerebral anoxia, is determined by the extent of the lesion, the location of the lesion, the nature of the lesion, and the degree of compensation of the cerebral circulation.

Extent of the lesion. It has been demonstrated both experimentally and clinically that with obstructing lesions of less than 50 per cent of luminal diameter there is minimal if any decrease of blood flow. With lesions greater than 50 per cent there is progressive diminution of blood flow through the vessel,[6] reaching the point of complete occlusion where the presence of distal run-off determines if a surgical approach is feasible.

The *location of the lesion* is another variable which determines the severity of symptoms, for a localized intracranial obstruction with little chance of collateral circulation may produce permanent damage, whereas a proximal lesion at the aortic arch is more likely to cause more diffuse temporary damage and any degree of dysfunction between these may be related to the site of multiple lesions in the cerebral vascular system. Location of the lesion is also important in deciding whether the lesion is surgically accessible.

The *nature of the lesion,* whether chronic or acute, is important in determining compensatory blood supply from the collateral circulation. A gradual subintimal obstructing process would allow maximal compensation of the collateral circulation. However, if the process involves intimal disruption and ulceration without significant obstruction, an embolus from this site may cause acute local ischemia for which the collateral circulation may not compensate.

The *degree of compensation of the cerebral collateral circulation* is the final pathway which determines the severity of symptoms. The brain receives blood from four main vessels, two carotids and two vertebrals, and when one or more of these vessels are partially or completely occluded one might expect symptoms corresponding to the obstructed vessel; however, clinically this is not the case. When one uses the concept of total cerebral blood flow, as is the case with extracranial vascular occlusion, it is understandable why some patients will have no symptoms, others will have symptoms of cerebral ischemia in relation to the obstructed vessels, while others will have symptoms referred from the region of the brain supplied by a patent vessel when the obstruction is at a different site. When one of the major vessels is obstructed, an increase in flow from the other patent cerebral vessels can compensate for the lack of blood flow to the potentially ischemic area. This can be ac-

complished through the circle of Willis, leptomeningeal arterial branches, external carotid sypplying the internal carotid, collaterals via the orbit, into the basilar arterial system by communication with the subclavian and anterior spinal arteries, the occipital branch of the external carotid with anastomosis to the vertebral artery as it enters the skull, the thyrocervical trunk, and ascending cervical arteries. It has been shown that patients with TIA have normal cerebral blood flow when asymptomatic, but with symptoms a decrease in total cerebral blood flow has been noted which is dependent on both the extracranial and intracranial collateral circulation.[3] As the cerebral blood flow becomes more dependent on the collateral circulation, any slight cerebral vascular insult as a decrease in cardiac output or fall in systemic blood pressure may precipitate symptoms.

Once the total cerebral circulatory pattern has been set, involving both the main channels and the collaterals, each individual is susceptible to a wide variety of symptoms; but what are the initiating factors which will cause symptoms? As alluded to previously, there are several factors:

Hypotension: Transient ischemic attacks are related to total cerebral blood flow. When an occlusive lesion of an extracranial vessel is present, the collateral circulation will dilate maximally and will become almost completely dependent on systemic blood pressure to maintain flow;[5,9] therefore, a fall in blood pressure can account for recurrence of symptoms from the same cerebral area for any individual patient. The variations of symptoms in many patients who have similar lesions can be explained by the dissimilarity of the collateral circulation.

Emboli of platelets, white blood cells, fat, and cholesterol from ulcerative or degenerative atheroma at the carotid bifurcation have been described in many patients with carotid artery stenosis and transient monocular blindness may be produced.[18,23,43] Emboli to the retinal artery have been seen to lodge at the bifurcation of the arteries, break up and move more distally, and ultimately disappear.[38] Although this surely plays a role in some patients with TIA, it does not account for recurrence of the same symptoms as is the usual case.

Other systemic factors which may precipitate TIA are a low cardiac output with its attendant decrease in cerebral blood flow, dehydration or polycythemia which increases stasis, and anemia which would decrease oxygenation of the cerebral cells.

ANGIOGRAPHY

Bilateral carotid angiography with anteroposterior and lateral views is performed generally for patients whose clinical picture suggests that stenosis of the carotid artery may be the cause of a TIA. Those patients whose clinical symptoms suggest vertebrobasilar insufficiency undergo left percutaneous carotid angiography and right retrograde brachial angiography to visualize both the right carotid and right vertebral arteries. If the carotids show no significant lesions, then four vessel study is completed with a retrograde left brachial injection to visualize the left

vertebral artery. In all patients the intracranial vascular bed is routinely visualized during angiography. Since the symptoms of cerebrovascular insufficiency are dependent on the collateral circulation, bilateral angiography affords the opportunity to evaluate the cerebral circulation if multiple lesions are present, as is the case in 75 per cent of patients with extracranial vascular disease. When bilateral internal carotid artery lesions which are amenable to surgery are present, the question arises which side should be done first? If angiography reveals a 90 per cent stenosis on one side and 50 per cent stenosis on the opposite side, greater benefits can be expected from operating on the more severe stenosis first. This will produce a greater increment of cerebral blood flow after surgery and will also provide a safer operative procedure.

Visualization of the intracranial vessels is helpful in several respects: (1) to evaluate cerebral atrophy in patients with intellectual impairment. (2) to observe intracranial mass lesions. (3) to evaluate vascular anomalies as arteriovenous malformation or aneurysms, and (4) to evaluate collateral circulation. This can be helpful in evaluating a patient who has bilateral internal carotid artery stenosis of the same degree when deciding which side should be repaired. If it can be determined whether there is cross filling, i.e., one carotid flow entering the opposite cranial hemisphere through the anterior communicating artery, this vessel is obviously carrying more of its share of cerebral blood flow and greater benefit might be expected if the opposite carotid artery was repaired first.

The site of lesions in patients with the aortic arch syndrome can usually be diagnosed by history and physical examination as described previously. Angiographic documentation by retrograde femoral, retrograde axillary, or brachial catheterization is readily done. These lesions are accessible and operable since both the proximal and the distal vessels are usually patent.

Bilateral carotid angiography is the mainstay in evaluating cerebrovascular insufficiency exclusive of the aortic arch syndrome.

There are four classes of patients:[33]

1. TIA with localizing signs of extracranial vascular disease.
2. TIA without physical evidence of a carotid lesion.
3. Bruits, palpable atheromatous masses at the carotid bifurcation, or pulse deficits in patients who do not have symptoms.
4. Generalized and progressive symptoms of cerebral vascular insufficiency in patients who, in addition, have TIA and physical signs of extracranial arterial lesions.

Patients with transient ischemic symptoms and a bruit over the carotid bifurcation comprise the largest single group (Class 1). In patients with carotid artery stenosis, there is a 75 per cent correlation between the bruit and angiographic evidence of an occlusive lesion.[27] In a high percentage of patients with vertebral artery stenosis there is an associated accessible lesion of the internal carotid artery.

The normal blood flow in each internal carotid artery is about 370 ml. per min., while in each vertebral artery the flow is about 45 ml. per min., so about 90 per cent[15, 37] of cerebral blood flow is accounted for by the internal carotid arteries. If one uses the concept of total cerebral blood flow

it is now obvious why detecting a carotid artery stenosis is so important in patients with symptomatic vertebral artery stenosis, for a significant lesion of the internal carotid artery, even without a bruit, can account for a more significant loss of cerebral blood flow.

Twenty per cent of patients with TIA and an accessible carotid lesion will have no physical evidence of a carotid artery lesion (Class 2). Although some of these arteries may be completely occluded, those patients with high grade stenosis may have no bruit and it would therefore be imperative to repair the artery before it completely obstructs.

Another group of patients that would fall in this category are those with kinking or rotational obstruction of the internal carotid or vertebral arteries. Those with internal carotid artery kinking are readily diagnosed with carotid angiography, however those patients with rotational obstruction of the vertebral artery may not be diagnosed with routine vertebral artery angiography unless cervical rotation is performed.

Asymptomatic patients with physical evidence of extracranial arterial occlusive disease such as a cervical bruit are studied if they are going to undergo major surgery.

INDICATIONS FOR SURGERY

Aortic arch syndrome. Patients with occlusive lesions involving the origins of the brachiocephalic arteries, when symptomatic, are candidates for surgical repair. An exception is asymptomatic stenosis of the origin of the left common carotid artery which can occasionally cause hemiplegia when it becomes completely occluded.[44]

Transient ischemic attacks when secondary to stenosis of the carotid bifurcation on one or both sides, with or without vertebral artery stenosis, kinked carotid arteries, intraluminal or extraluminal obstruction of the vertebral arteries, or angiographic evidence of an ulcerative lesion with or without significant stenosis are candidates for surgical repair. Complete occlusion of the internal carotid artery is not considered an operable lesion.

Progressing stroke patients are not considered surgical candidates except in the following instances: (1) neurologic deficits appearing following angiography where a subintimal hematoma or dissection has occurred; (2) a deficit appearing in the postoperative period following a successful carotid endarterectomy; and (3) a previously audible bruit is lost, or when a stroke during postangiographic period appears after the demonstration of a severely stenotic lesion. These patients are taken to surgery within the hour.

Patients with *completed strokes* are not candidates for surgical repair unless associated with TIA.

Treatment for *asymptomatic lesions* of the carotid bifurcation is controversial. Javid,[22] in a study of the natural history of carotid bifurcation atheroma, showed the lesion to progress in 60 per cent of the patients studied during a 1 to 9 year follow-up, 40 per cent of these requiring surgical intervention (Fig. 1). Other reports[41, 44] showed a 30 per cent incidence of cerebral infarction during a two year follow-up in patients with a stenosis of the internal carotid artery greater than 50 per cent, while

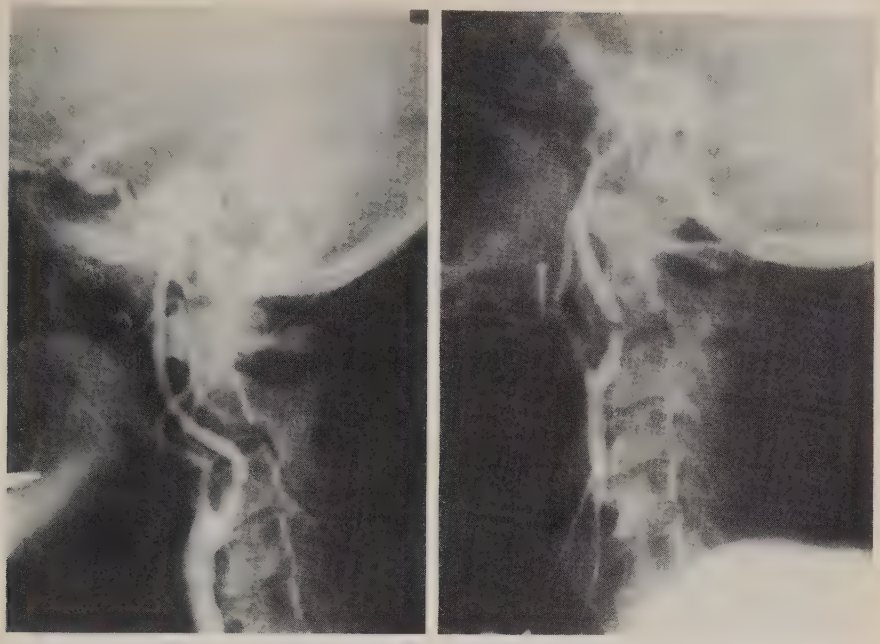

Figure 1. Progression of a carotid lesion in 1 year. After the second angiogram on the right, emergency carotid endarterectomy was done.

others refute this, showing a 3 per cent incidence of strokes during the same period.[27] However, since the stroke potential of these lesions is not known, and about 75 per cent will have TIA before a cerebral catastrophe, it has been the policy at this institution to repair asymptomatic carotid artery lesions only before a major surgical procedure during which a fall in blood pressure may precipitate thrombosis of a compromised carotid artery.

RESULTS OF SURGERY

The surgical techniques used have been described in detail in other publications.[24, 25] In general, endarterectomy of the carotid bifurcation has been done using a temporary shunt. Vertebral artery stenosis has been treated utilizing endarterectomy and vein patch. Left subclavian occlusion is treated by a carotid-subclavian graft through a cervical approach. Occlusions of the aortic arch branches other than the left subclavian are bypassed from the aortic arch to the distal open vessel.

Surgical results are as follows: In 1100 cases of TIA with carotid artery stenosis, surgery resulted in an asymptomatic patient in 80 to 90 per cent of cases with a stroke incidence of 1.8 per cent and a surgical mortality of 2 per cent.[7, 8, 10, 41] Nonsurgical treatment of these resulted in a stroke incidence of 10 to 27 per cent.[14, 40, 41] Carotid artery surgery for patients with vertebral artery obstruction and vertebrobasilar insufficiency with concomitant carotid artery stenosis results in 60 to 90 per cent improvement with complete relief in two thirds of these patients.[23, 30]

TIA with vertebral artery stenosis of either the intraluminal or extraluminal type results in 90 per cent improvement.[8, 20, 36]

Progressing strokes which have followed cerebral angiography or carotid endarterectomy are considered for surgery. Fifteen such patients have undergone emergency carotid surgery for progressive neurologic deficits following cerebral angiography; of these, 8 improved, 2 of which went on to complete recovery, 6 did not improve and 1 died. Emergency surgery was done in 7 patients who had a stroke in the immediate postoperative period following carotid endarterectomy; of these, 3 improved, 2 of which totally recovered, 2 were unimproved and 1 died.[34]

The surgical treatment of *completed strokes* holds no advantage over medical therapy; following surgery 35 per cent improved, 20 to 30 per cent were unchanged, 10 to 20 per cent became worse, and 15 to 30 per cent died.[13, 30]

CONCLUSION

Surgery to relieve extracranial obstruction to cerebral flow has proved to be a valid treatment for cerebral vascular insufficiency. This is particularly true of isolated stenosis of carotid bifurcation producing transient ischemic attacks.

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Orthognathic Surgery

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Orthognathic surgery was performed by Hullihen[22] in 1849 to correct an open bite. Yet little was written concerning orthognathic surgery for the next 50 years. This specialized type of surgery was practiced primarily in Europe until Obwegeser of Zurich gave his lectures in Washington, D.C., in 1966. Since that time orthognathic surgery has been widely performed in America.

Orthognathic surgery is not a replacement for orthodontia. Whenever possible the conservative approach of orthodontia should be used. However, there are reasons for using the surgical approach: (1) when the malocclusion is of such a magnitude that orthodontia alone cannot correct the deficiency; (2) when the individual is not willing to wear "braces" for 1 to 2 years but does want correction of a gross deformity.

DIAGNOSIS AND WORK-UP

Occlusal disharmonies may result from disease or trauma, or they may be developmental. The analysis and work-up of malocclusions require the skills derived from orthodontia, prosthodontia, and surgery. The repositioning of the jaws for functional and pleasing aesthetics is the goal.

The clinician who treats malocclusion should have a knowledge of the occlusal mechanism, namely, the anatomic structure of the teeth, bone, and muscles of mastication, and their relationship to balanced functional occlusion. The functional anatomy of the temporomandibular joint is also involved in relation to centric occlusion. Therefore, the clinician should be able to predict the functional as well as the aesthetic results.

It is important that a patient desire orthognathic surgery. He should be psychologically sound and physically fit. He should be aware of the few days of postsurgical pain and the inconvenience of 6 weeks of intermaxillary fixation.

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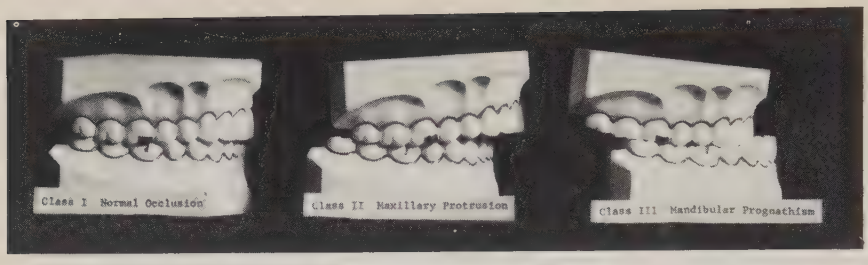

Figure 1. Basic occlusion patterns: Class I normal molar occlusion, Class II maxillary protrusion, Class III mandibular prognathism.

Three modalities considered necessary by most operators today in treating malocclusions are clinical evaluation, cephalometric analysis, and model study.

Clinical Evaluation

Clinical evaluation refers to the assessment of the general appearance of the patient, as seen by himself, his acquaintances, and the surgeon. The patient may have a "Dick Tracy" jaw, an "Andy Gump" chin, an open bite, or "buck teeth," or he may be unable to open his jaw. The patient is usually quite aware of his facial profile and the way he bites; however, he is often unaware that such a defect can be corrected.

Functional malocclusion was classified by E. H. Angle[2] in 1899 into three basic categories, which are used today with only slight modifications: Class I, normal molar occlusion; Class II, maxillary protrusion; and Class III, mandibular prognathism (Fig. 1). In Class I malocclusion the teeth are in good interdigitation. The upper incisors just overlap the labial surface of the lower incisors and the mesiobuccal cusp of the upper first molar digitates into the buccal central groove of the lower first molar, with only slight malpositioning of one or more of the anterior teeth. A Class II malocclusion is present if the lower first molar is posterior to the upper first molar. This is usually caused by a maxillary protrusion but may be the result of mandibular retrusion. A Class III malocclusion is evident if the lower first molar is anterior to the upper first molar. This is usually caused by a mandibular protrusion but may be caused by maxillary retrusion (pseudoprognathism).

Cephalometrics

Cephalometrics is a radiographic system used traditionally by orthodontists and anthropologists to study the relationship and growth patterns of the face and cranium. Many investigators have contributed to our understanding of craniofacial measurements.[16, 29, 30, 34, 50]

We have derived a workable method for surgery from a variety of orthodontic cephalometric systems. Our analysis is shown in Figure 2*A*.

This scheme of analysis is intended to assist in evaluating craniofacial measurements in one dimension, namely the lateral view. Analysis of horizontal and unilateral anomalies are determined principally by clinical and radiographic evidence. The radiographic points of reference shown in Figure 2*B* and *C* are:

PATIENT: Female AGE: 18 DATE: 6-71

RELATIONSHIP	MEASUREMENT	RANGE	MEAN	PRE-OPERATIVE	INTERPRETATION	POST-OPERATIVE
MAXILLA to CRANIUM	SNA	79-85°	82°	90°	Maxillary Prognathia	83°
MANDIBLE to CRANIUM	SNB	76-84°	80°	81°	WNL	78°
MANDIBLE to MAXILLA	ANB	-1-+5°	+2°	+9°	Wide discrepancy Mandible to Maxilla	+5°
OPEN OR CLOSED BITE TENDENCY	SN-G_oG_n	30-35°	33°	33°	WNL	33°
AXIAL INCLINATION OF TEETH	UI-NA	18-32°	25°	22°	WNL	26°
	LI-NB	16-30°	23°	47°	Mandibular Alveolar Prognathia	42°
CHIN BUTTON	Pg-NB	0-4mm	2mm	-2mm	WNL	+3mm
FACIAL PROPORTIONS	NA:APg		1:1	56:58	WNL	57:60
FACIAL PROFILE	FH-ChinLips	75°-85°	80°	60°	Protrusive lower face	75°

DIAGNOSIS Maxillary prognathia with mandibular alveolar prognathia

RECOMMENDATIONS

1. Anterior maxillary osteotomy to correct maxillary prognathia
2. Anterior mandibular segmental osteotomy to correct mandibular alveolar prognathia
3. Extracted right and left maxillary second bicuspids
   Extracted right first bicuspid and left second bicuspid in the mandible

A

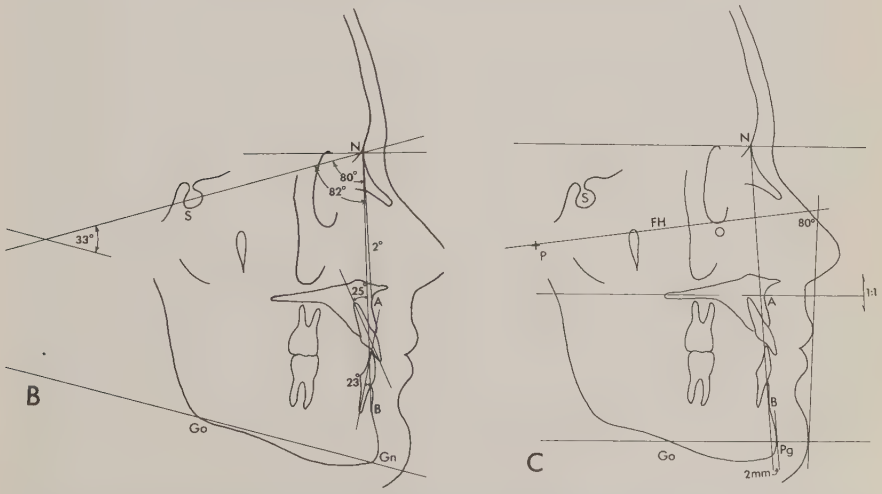

Figure 2. *A*, Cephalometric analysis form used by the oral surgery section of the University of Chicago Hospitals and Clinics. An analysis of Case 6 is used as a demonstration. *B*, Line drawing showing the following normal cephalometric values SNA, SNB, ANB, SNGoGN, UI-NA and LI-NB. *C*, Cephalometric values Pg-NB, NA:APg and FH-Chin Lips angle. (See text, page 256, for explanation of abbreviations.)

S, Sella: the midpoint of the sella turcica.

N, Nasion: the depression at the junction of the frontonasal suture.

A, Subspinale: the midline depression between the anterior nasal spine and alveolar process of the maxilla.

B, Supramentale: the midline depression on the anterior surface of the mandible.

Go, Gonion: the most posterior inferior point at the angle of the mandible.

Gn, Gnathion: the most anterior inferior point in the midline of the mandible.

UI, Upper incisor: the axis from the incisal tip to the apex of the upper central incision tooth.

LI, Lower incisor: the axis from the incisal tip to the apex of the lower central incisor tooth.

Pg, Pogonion: the most anterior point of the bony chin.

P, Porion: the upper edge of the bony auditory meatus.

O, Orbital: the lower point on the margin of the orbit.

FH, Frankfort horizontal: A line connecting the porion and the orbitale.

SN, Sella nasion: A line, identified on a lateral cephalogram, connecting sella and nasion. Because of its stability after early growth, it provides a constant reference line.

SNA angle: indicates the anterior or posterior placement of the maxilla along the sella nasion or cranium.

SNB angle: indicates the anterior or posterior placement of the mandible along the sella nasion.

ANB angle: shows the anterior posterior relationship of the mandible to the maxilla. Usually the maxilla is a few degrees anterior to the mandible.

SN-GoGn angle: indicates the degree of open or closed bite.

UI-NA angle: indicates the axial inclination of the upper incisor teeth.

LI-NB angle: indicates the axial inclination of the lower incisor teeth.

Pg-NB distance: indicates the degree of chin prominence relative to the rest of the mandible.

NA : A Pg: gives the relative height of the midface to the lower face. If the face has an NA:A Pg ratio of about 1:1, correction of orthognathic disharmonies must be done with caution so as not to change this normal ratio.

FH-Chin Lip angle: the angle formed at the junction of the Frankfort horizontal (P-O) and chin lip line correlates with the soft tissue profile. A radiopaque media placed on the profile prior to taking the radiograph will clearly define the soft tissue profile.

This cephalometric analysis is derived from "normal" Caucasians and it must be applied with caution to different cases. Orientals have a gentle flattened profile, while Negroes tend toward a protrusive profile. As a general rule cephalometric analysis is a practical and useful tool in determining the craniofacial relationships. Clinical impression and model study without cephalometrics may cause one to reach a wrong diagnosis, as demonstrated in Cases 3 and 7 of this study. There are some patients, however, who show little or no correlation between an abnormal cephalogram and an acceptable and sometimes aesthetically pleasing soft tissue profile. In this event the cephalometrics are disregarded in favor of the pleasing profile. A cephalometric protracter which enables one to read these line angles directly from radiographs or tracings is available from any dental supply company.

Models

The ideas resulting from the correlation of cephalometric studies and clinical judgment are worked out on plaster models (Figs. 7*C*, 8*E*, 11*C*, 14*A*, and 15*C*). We routinely have our prosthetic laboratory pour 6 sets of the original dental models in a mold with a silicone rubber base, using a

fast acting catalyst. This mold is kept and any number of additional models can be poured from it. It is on the manipulation and sectioning of the models that the limits of correction and the details of function are determined. After the desired new occlusion has been achieved on the models, they are mounted in centric occlusion. The corrected set-up is then compared with the original set-up and the differences noted. The differences dictate the location and extent of surgery to be performed. At times a compromise has to be made among several variables such as ideal surgery, functional occlusion, and aesthetics.

Stabilization of the new occlusion may be accomplished in a variety of ways, depending on the type of case. Full orthodontic banding may be done preoperatively or postoperatively for stabilization of tooth movements. Standard arch bars, cast metallic arch bars, cast occlusal splints, and acrylic splints all may be used to stabilize the new occlusion produced by surgery. If an entire maxilla or mandible is to be moved as a unit, stabilization splints may be placed preoperatively. However, if the maxilla or mandible is to be sectioned, the splints must be placed after surgery. An exception to this is when sectional splints are used. In this case part of the splint can be seated preoperatively and the second part bolted in position at the time of operation.

CASE REPORTS

Eight cases are presented to illustrate various types of malocclusion and their treatment.

Case 1. Class I Malocclusion

Class I malocclusion may be defined as normal molar occlusion with minor discrepancies in tooth position within the dental arch. This 19 year old girl came to the clinic with a relatively normal soft tissue profile but spaced, protruding lower anterior teeth (Fig. 3*A*). The discrepancy was diagnosed as being primarily in the anterior alveolar portion of the maxilla and mandible. The axial inclination of the upper incisors was 38°, the lower incisors 40°. Because of the normal profile and satisfactory molar occlusion, it was decided to treat only the mandibular anterior segment. This patient could have been treated orthodontically; however, she would not accept prolonged treatment. Corticotomy with a prefabricated removable orthodontic appliance was planned (Fig. 3*B*).

The anterior mandible was degloved intraorally. The alveolar cortex alone was sectioned carefully with a fine drill between each tooth so as not to disrupt the periodontal membrane of the root or the blood supply of the medullary bone. A horizontal cut below the apices of the tooth was made on the anterior and posterior side of the teeth to be moved (Fig. 3*C*).

Figure 3*D* and 3*E* shows the mucoperiosteum repositioned, sutured, and secured with a periodontal pack. The appliance was then activated and inserted, and caused the mandibular teeth to move into an edge to edge bite after only 4 weeks of orthodontic force (Fig. 3*F*).

Single or multiple teeth may be repositioned in this manner. Normal treatment usually takes 6 to 12 weeks, then a passive retainer should be

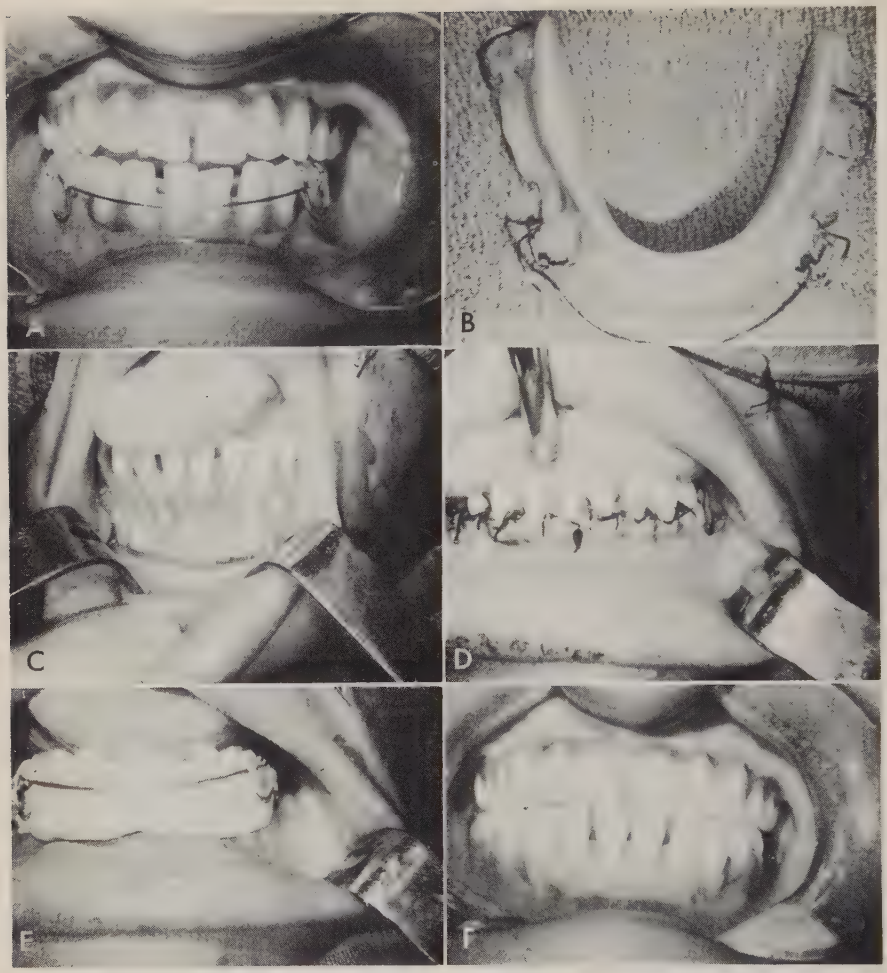

Figure 3. (Case 1). Class I malocclusion. *A*, Mandibular teeth splayed in protrusion. *B*, Removable orthodontic appliance. *C*, Corticotomy cuts on mandible. *D*, Mucoperiosteal flap sutured. *E*, Periodontal pack in position. *F*, Four weeks after operation.

worn for 7 to 15 months. According to Köle and others[15, 25] radiographic evidence shows no postoperative root resorption. Tooth vitality tests after 6 weeks are always positive, and there is no pocket formation. The liability to relapse is less when the supporting bone is moved with the tooth.

Case 2. Mandibular Prognathism

This 20 year old woman presents with an obvious "Dick Tracy" chin and a Class III malocclusion (Fig. 4*A*, *C*, and *E*). SNB is 85°, ANB –2°, and facial profile 85°. When comparing these measurements with the normal cephalometric values given in Figure 2*A*, and after clinical evaluation of her models and soft tissue profile, mandibular prognathism was diag-

nosed. Bilateral vertical osteotomies were performed through bilateral submandibular approaches. The posterior rami and condyles have been approached by operators in a variety of ways.[1, 7, 10, 12, 20, 42, 44, 46-48] As previously determined on the models, the teeth were placed into occlusion and maintained with arch bars and intermaxillary fixation for 6 weeks. Postoperative views of the teeth and profile are shown (Fig. 4*B*, *D*, and *F*).

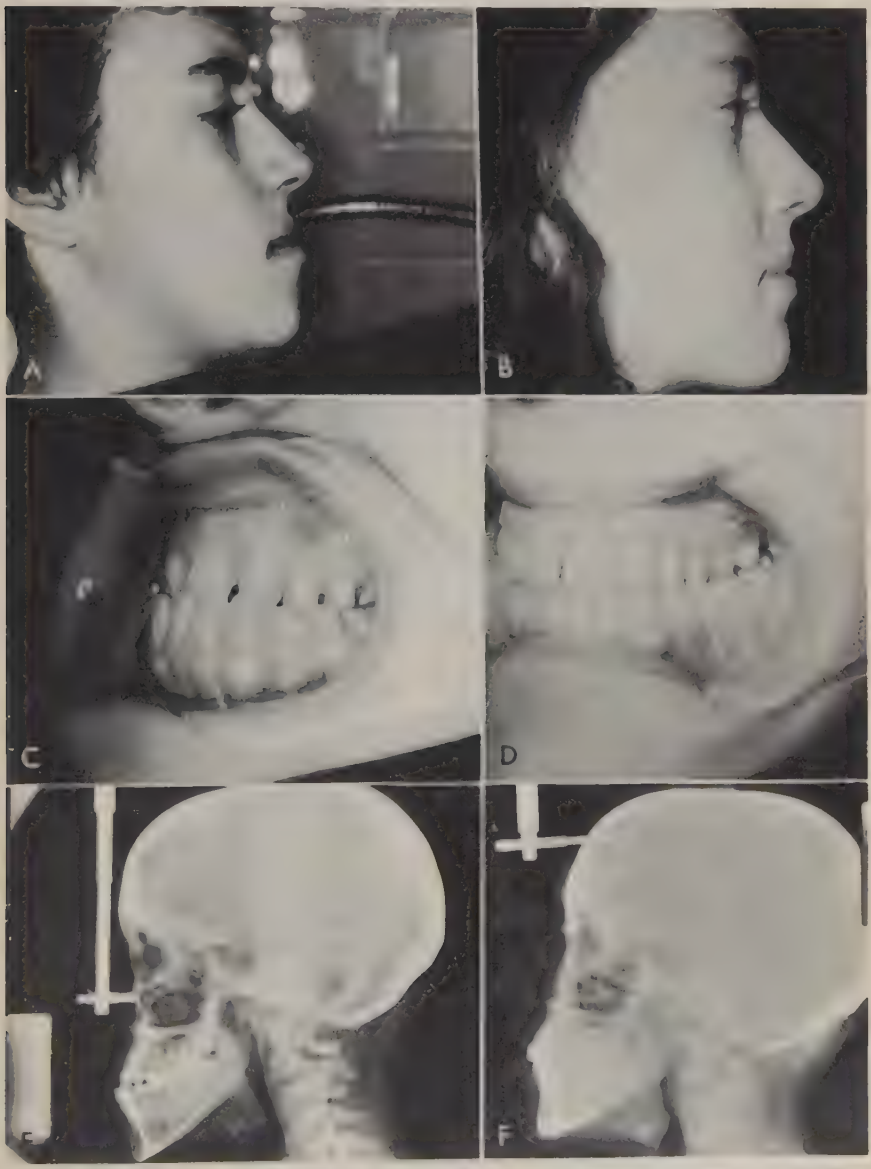

Figure 4. (Case 2) Mandibular prognathism. *A*, Clinical prognathic mandible. *C*, Class III malocclusion. *E*, Lateral cephalometric radiograph showing protrusion of mandible. *B*,*D*, *F*, Evidence of surgical correction of a class III malocclusion.

Case 3. Mandibular Prognathism with Crossbite

At first impression the third patient, a 26 year old woman, has a problem similar to that of patient 2 (Fig. 5*A*). However, when looking at the occlusion there is an obvious crossbite in addition to the Class III malocclusion (Fig. 5*C*). Cephalometric analysis reveals a normal mandible to cranium relationship, with an SNB of 80°, but abnormal maxilla cranium relationship, with an SNA of 78°. Therefore, this case presents as a slightly retropositioned maxilla which exaggerates a normal mandible. Considering the cephalometric analysis facial profile and model study, a pseudoprognathic mandible was diagnosed. However, retropositioning the mandible the minimal amount necessary to correct the malocclusion would still provide a pleasing profile in this case. Therefore, bilateral vertical sliding osteotomies were performed, as in the first patient, allowing correction of the Class III malocclusion as well as the crossbite. A lateral view is illustrated (Fig. 5*E*). Postoperative photographs of the occlusion and profile are shown in Figure 5*B* and *D*.

Case 4. Mandibular Prognathism with Macrogenia

This 20 year old woman impresses one as having a long face and a prominent chin (Fig. 6*A*, *C*, and *E*). Inspection of the occlusion reveals many missing teeth and protrusion of the anterior mandibular teeth (Fig. 7*A*). Cephalometric readings: SNB 84°, ANB —4° and facial proportions of 65:70 mm. substantiate this impression. Because of the extensive loss of maxillary teeth and poor condition of several remaining teeth, all the maxillary teeth and the molar teeth in the mandible were removed. The models were mounted in centric occlusion and temporary maxillary and mandibular splints were constructed to fit the new occlusion (Fig. 7*C*). To correct this malocclusion and facial abnormality, bilateral vertical osteotomies and reduction genioplasty were planned using a retromandibular and intraoral approach.

The genioplasty is performed by first degloving the mucoperiosteum on the anterior position of the mandible. Care is taken not to sever the mental nerves. A 6 mm. wedge of bone is then removed with a fine bur (Fig. 7*D* and *F*). The inferior border of the mandible is raised into the defect and stabilized with stainless steel wire. Modifications of this procedure have been described by several writers.[20] The mucoperiosteum is then repositioned and sutured.

In making the retromandibular approach to the condyle an incision is made 1 cm. below the bony ear canal and 1 cm. posterior to the ramus (Fig. 7*E*). After incising the skin, parotid capsule, and parotid gland one must then turn anteriorly at right angles and by blunt dissection cut through the fibrous connective tissue to the posterior border of the ramus. At the ramus, further blunt dissection will expose the condyle and mandibular notch where the vertical osteotomy is performed. The facial nerve and maxillary artery must be avoided. The incision sites are covered with wet sterile dressings, and maxillary and mandibular splints are then inserted and stabilized using stainless steel maxillary perialveolar and circum-mandibular wires (Fig. 6*F*). The incision sites are uncovered and the condyles checked for proper position.

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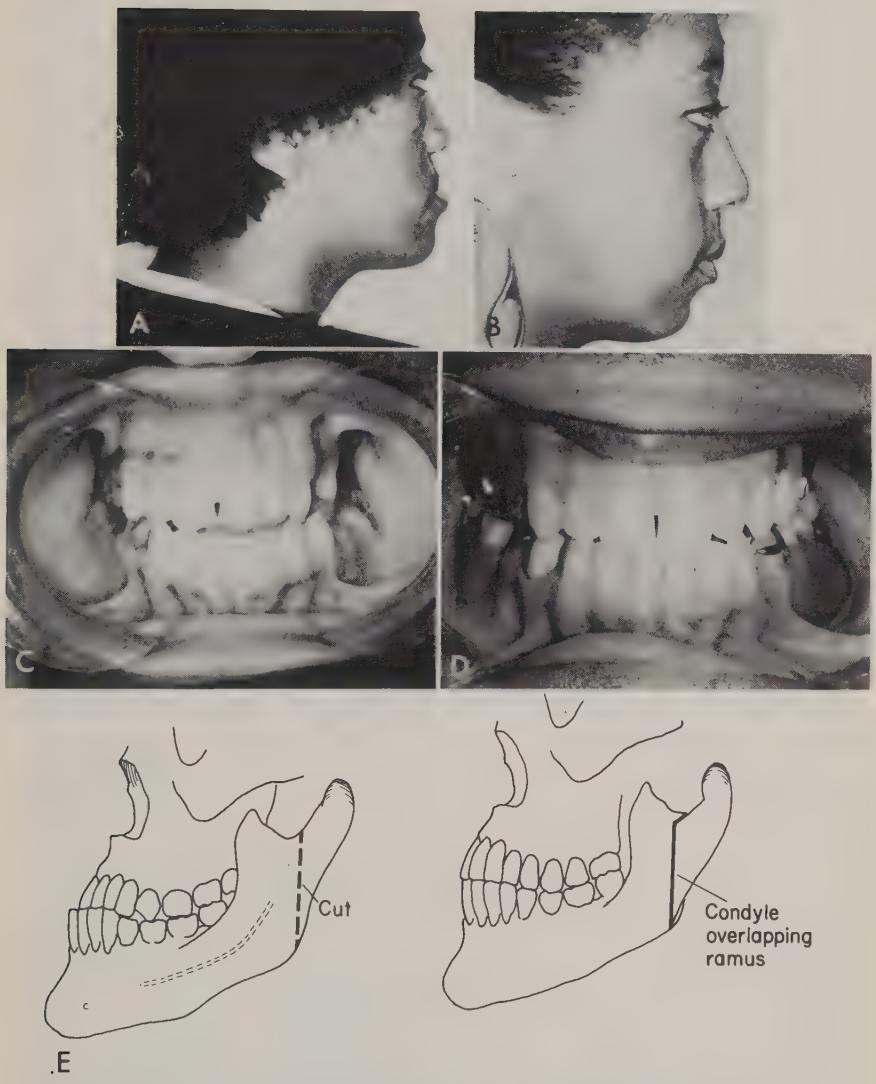

Figure 5. (Case 3). Mandibular prognathism with crossbite. *A*, Clinical prognathia of the mandible. *C*, Class III malocclusion with a crossbite. *B*, *D*, Postoperative appearance of the profile and occlusion. *E*, Drawing of a mandible showing a bilateral vertical sliding osteotomy cut with posterior positioning of the body.

Figure 6. (Case 4). Mandibular prognathism with macrogenia. *A*, *C*, *E*, A patient with a prognathic mandible and macrogenia. *B*, *D*, *F*, Clinical and radiographic profile after surgery.

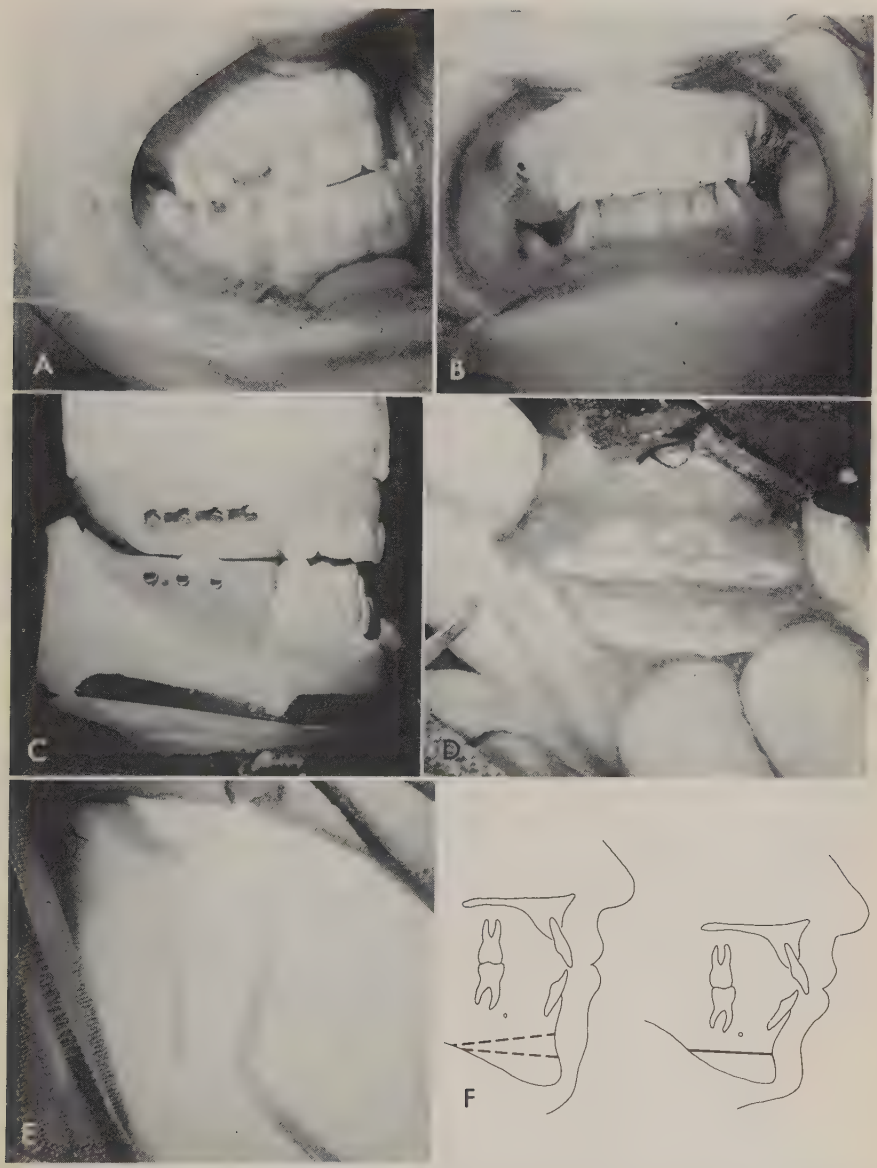

Figure 7. (Case 4). Mandibular prognathism with macrogenia. *A*, An occlusal view showing several decayed teeth and a Class III malocclusion. *C*, Corrected bite with temporary splints on model. *B*, Temporary splint positioned in mouth after mandibular osteotomies and removal of teeth. *E*, Skin lines painted preoperatively for retromandibular approach to the condyles. *D, F*, Surgical cuts and drawings of genioplasty.

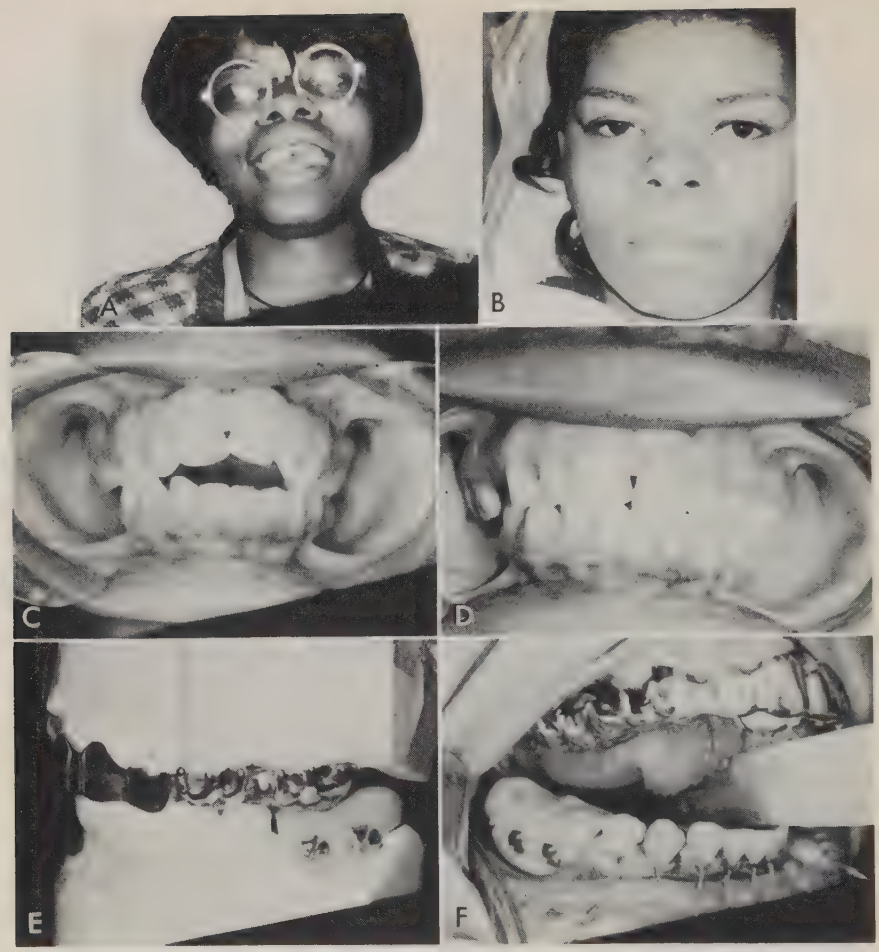

Figure 8. (Case 5). Maxillary anterior open bite. *A, C,* Class II malocclusion with an anterior open bite. *B, D,* Postoperative appearance of patient with corrected anterior open bite. *E,* Cast maxillary splint and removable mandibular splint on the adjusted model. *F,* Splints cemented to the surgically corrected maxilla and wired to the mandible.

The incisions are closed taking extra precaution to make a right closure of the parotid capsule, precluding the possibility of a parotid fistula. The extraoral and intraoral sutures are removed in 7 days, while the maxillary and mandibular splints remain in position for 6 weeks.

Postoperatively the patient wore the upper splint as a denture while waiting to have full dentures constructed (Fig. 7*B*). Postoperatively the cephalometric readings show a change of SNB 84° to 78°, and ANB from −4° to 2°. The occlusion is improved and the profile is pleasing (Fig. 6*B* and *D*, Fig. 7*B*).

Case 5. Maxillary Anterior Open Bite

This 25 year old woman presents with a severe anterior open bite (Fig. 8*A* and *C*). Cephalometric studies were normal except for a pro-

trusive maxilla registering an SNA of 91°. Models were cut, the right and left maxillary first bicuspid removed, and the models mounted in the new occlusion. Duplicate casting models were made for the construction of an upper all cast crown splint and a lower cast removable partial splint (Fig. 8*E*).

Surgery, as determined by exact model cuts, involved extracting the maxillary first bicuspids and removing a vertical strip of bone the same width on the right and left lateral surfaces of the maxillary. The cut extended above the apices of the cuspid teeth (Fig. 9*A* and *C*). These vertical cuts were connected anteriorly via a tunnel beneath the mucoperiosteum to the midline of the maxilla above the apices of the teeth. A palatal flap was made and a section of palatal bone removed from the maxilla, connecting the extraction sites. The anterior palatal fragment is now entirely free except for its attachment to the vomer. The maxilla and vomer are separated using a fine osteotome via an intraoral nasal approach. Finally, the free floating segment is sectioned at the midline in an anterior posterior direction (Fig. 9*B* and *D*). The anterior maxillary segments are gently repositioned and the palatal and buccal flaps sutured. The preformed cast splints are then cemented onto the upper teeth and wired to the lower teeth (Fig. 8*F*). The safety of this procedure has been verified by Bell[5] in revascularization studies on monkeys. One year postoperatively the patient can close her lips without tension (Fig. 8*B*). The SNA angle was reduced from 91° to 84°. The upper right central incisor could be further straightened with orthodontic banding; however, the patient is satisfied with her occlusion (Fig. 8*D*).

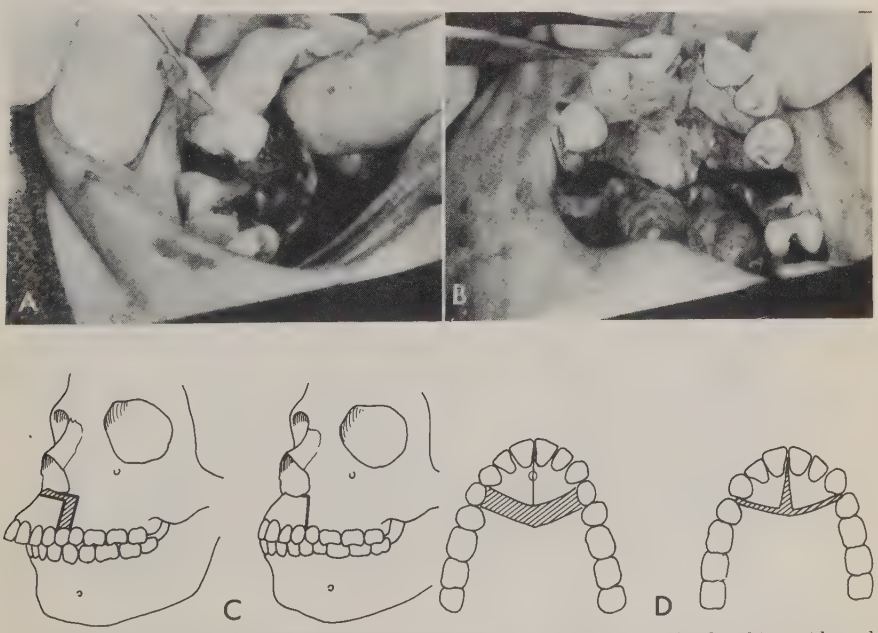

Figure 9. (Case 5). Maxillary anterior open bite. *A*, *C*, Removal of the first bicuspids and lateral maxillary bone wedge resection followed by retropositioning of the anterior maxillary segment. *B*, *D*, Palatal cut and midline split of anterior maxilla.

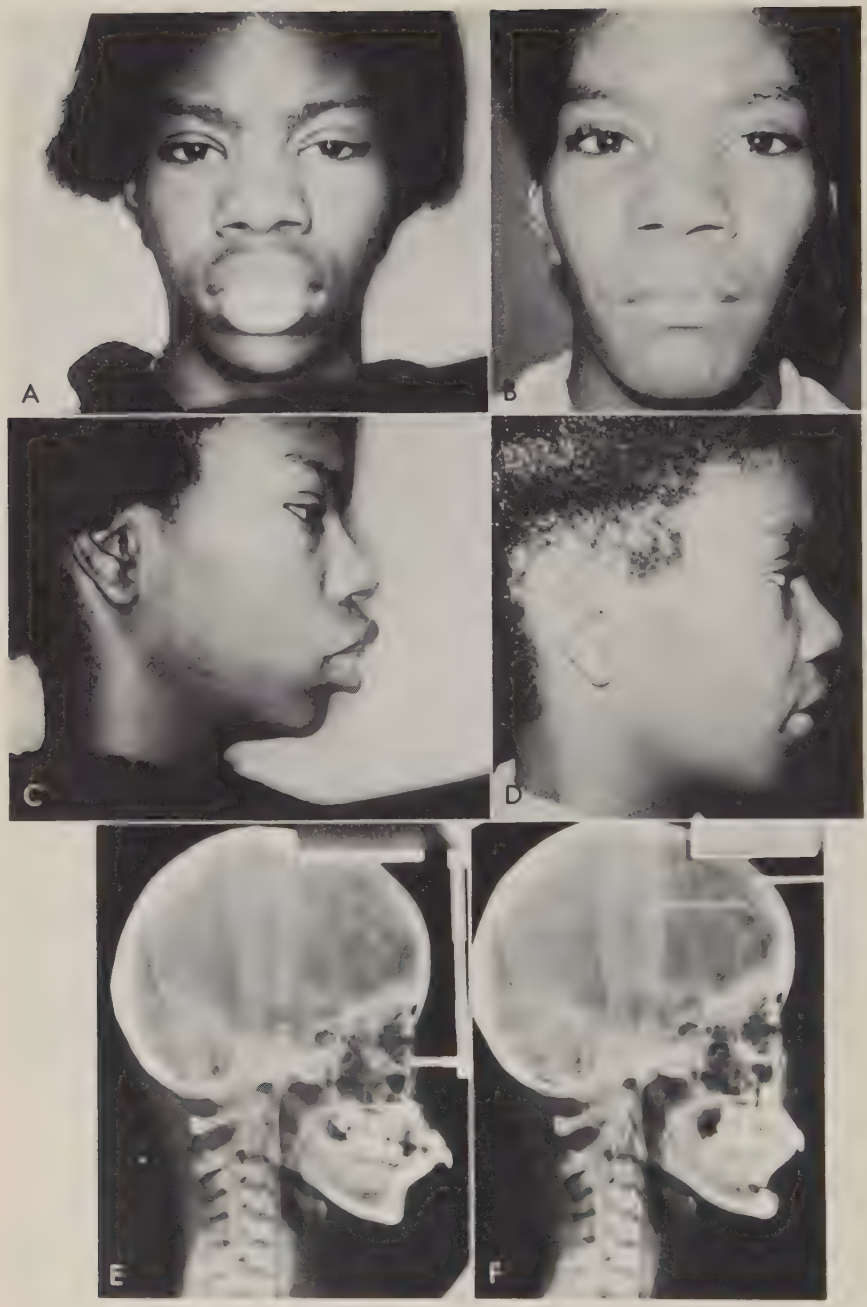

Figure 10. (Case 6). Bimaxillary anterior prognathia. *A, C, E,* Bimaxillary protrusion giving facial, profile and lateral radiographic views. *B, D, F.* Appearance of the patient after surgical correction of bimaxillary protrusion.

Case 6. Bimaxillary Anterior Prognathia

This 18 year old female was referred to our clinic by the orthodontic department. She was shy and introverted. She presented with gross "buck teeth" and swollen gingiva (Fig. 10*A*, *C*, and *E*, 11*A*). The gingivitis was aggravated by poor oral hygiene, mouth breathing, and inability to close the lips. The cephalometrics of this case are given in Figure 2*A*. There is maxillary prognathia with an SNA of 90°, an overjet of the maxilla in relation to the mandible ANB 9°, and an extreme mandibular alveolar protrusion of LI-NB 47°. The facial profile FH-chin lips is 60°, also indicating a protrusive lower face. After correlating our clinical impression, the cephalometrics, and model study, it was decided to remove the four bicuspids and retroposition the anterior maxilla and anterior mandible 5 mm. This was performed on the models, then maxillary and mandibular splints were constructed for this new bite (Fig. 11*C*).* Surgery involved removing the mandibular right first bicuspid and left second bicuspid, and removing a vertical block of bone at the extraction sites. The mucoperiosteum on the anterior mandible was degloved and the alveolus sectioned horizontally below the apices of the teeth from right to left bicuspids. The depth of the horizontal cut was predetermined by radiographic study (Figs. 10*E*, 11*E*, 12). The mental nerves were isolated and protected during the osteotomy. The fragment was mobilized, the mucoperiosteum sutured, and the preformed acrylic splint wired into position.

After removing the maxillary second bicuspids an anterior maxillary osteotomy was performed (Figs. 11*F* and 12). The maxillary acrylic splint was inserted and the teeth were brought into occlusion (Fig. 11*D*). Fixation was maintained for 6 weeks. Postoperative cephalometrics reveal that preoperative readings are now nearly all within normal limits (Fig. 2*A*). There was a remarkable improvement in the patient's personality postoperatively and she was eager to be seen by the other doctors on grand rounds where her case was presented. A photograph taken 1 year later shows that the patient is controlling her periodontal disease and is maintaining stable occlusion (Fig. 11*B*). Postoperative photographs reveal an improved facial profile (Fig. 10*B*, *D*, and *F*).

Case 7. Complete Bimaxillary Retrognathia

This 23 year old woman stated that as a young girl she had had a small chin and had been treated for malocclusion by an orthodontist who removed 4 bicuspid teeth and produced a perfect occlusion. The difficulty was however, that the retrusive chin already present was amplified (Fig. 13*A*). She was conscious of this condition for years and often went around jutting out her lower jaw to improve her appearance. When the patient protruded her lower jaw, she had a normal profile; however, this would put her into Class III malocclusion, and was untenable as a permanent position. Cephalometric readings were SNA 74° and SNB 67°. The final analysis revealed a combined maxillary and mandibular retrognathia. This craniofacial disharmony could not be corrected with a chin

*Several authors (references 3, 4, 24, 26, 32, 33, 35, 37, 40) have described procedures for repositioning posterior alveolar segments as well as anterior segments, similar to our case.

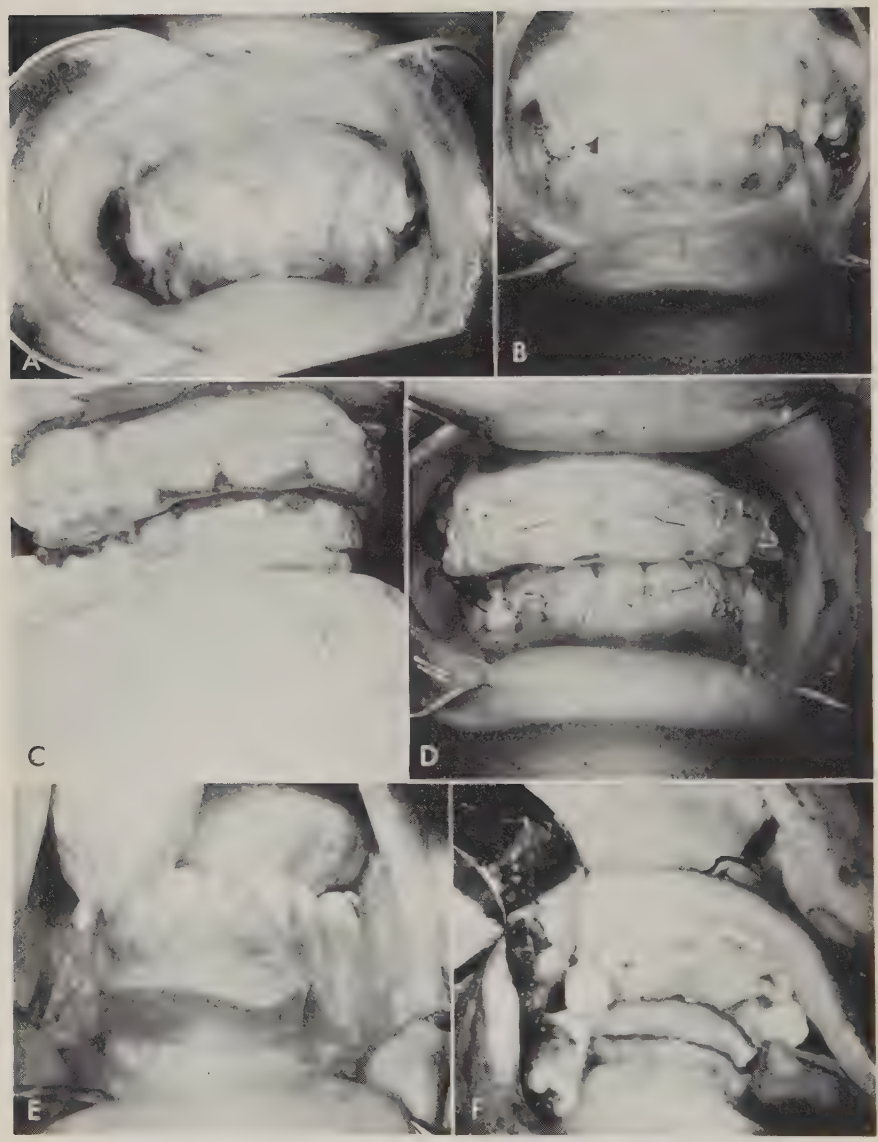

Figure 11. (Case 6). Bimaxillary anterior prognathia. *A*, *B*, The inflamed gingiva contrasted with healthy gingiva when the patient could close her lips after surgery. *C*. *D*. Acrylic splints mounted on the modified models and then wired to maxilla and mandible. *E*, Drill holes marking area of mandibular cut below the apices of the teeth, predetermined by radiographs. *F*, Palatal cut and removal of two bicuspids.

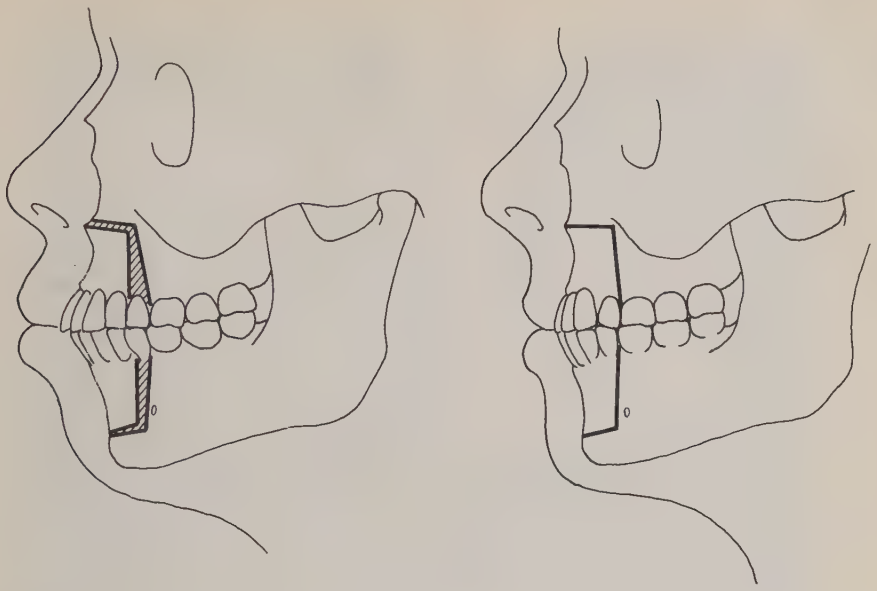

Figure 12. Drawing of a lateral view for surgery and repositioning of bimaxillary protrusion as performed in the sixth case.

implant for it would not improve the sagging neckline but rather give a "C" look to her face. Complete anterior reposition of the maxilla and mandible was decided as the treatment of choice. The procedure was done in two stages, first the mandible, then at a later date the maxilla was moved forward. Models were mounted in mandibular protrusion at the desired distance, and then an acrylic splint and cast arch bars were constructed (Fig. 14*A*).

The mandible was sectioned bilaterally using the modified intraoral technique of Obwegeser[6, 11, 23, 36, 43] and was advanced forward 10 mm. (Fig. 14*B*). After 6 weeks in intermaxillary fixation, the patient was allowed to function with her Class III bite for 1 week (Fig. 13*C* and *D*). The second stage operation, a complete maxillary osteotomy, similar to a LeFort I fracture, was then performed, as described below.

An incision is made in the maxillary vestibule from right to left molar areas. Hemostasis is accomplished. A small osteotome is used to section the maxilla above the apices of the teeth from the right to the left pterygoid plates (Fig. 14*D*). Cuts are made distally along the lateral wall of the nose and the vomer is sectioned in the midline at the same level as the lateral cuts. A large curved chisel is placed at the distal surface of the maxillary tuberosity and force is directed from a lateral to a medial direction, sectioning the bony union of the maxilla and pterygoid plate. The procedure is performed on both sides of the maxilla. The chisel must be kept low to avoid the descending palatine artery and pterygoid venous plexus (Fig. 14*C* and *D*). The maxilla is now completely free except for a small portion of the lateral nasal wall in the posterior region, the vertical plates of the palatine bones. Two disimpaction forceps are placed in each nostril and onto the palate, then with gentle steady pressure the posterior

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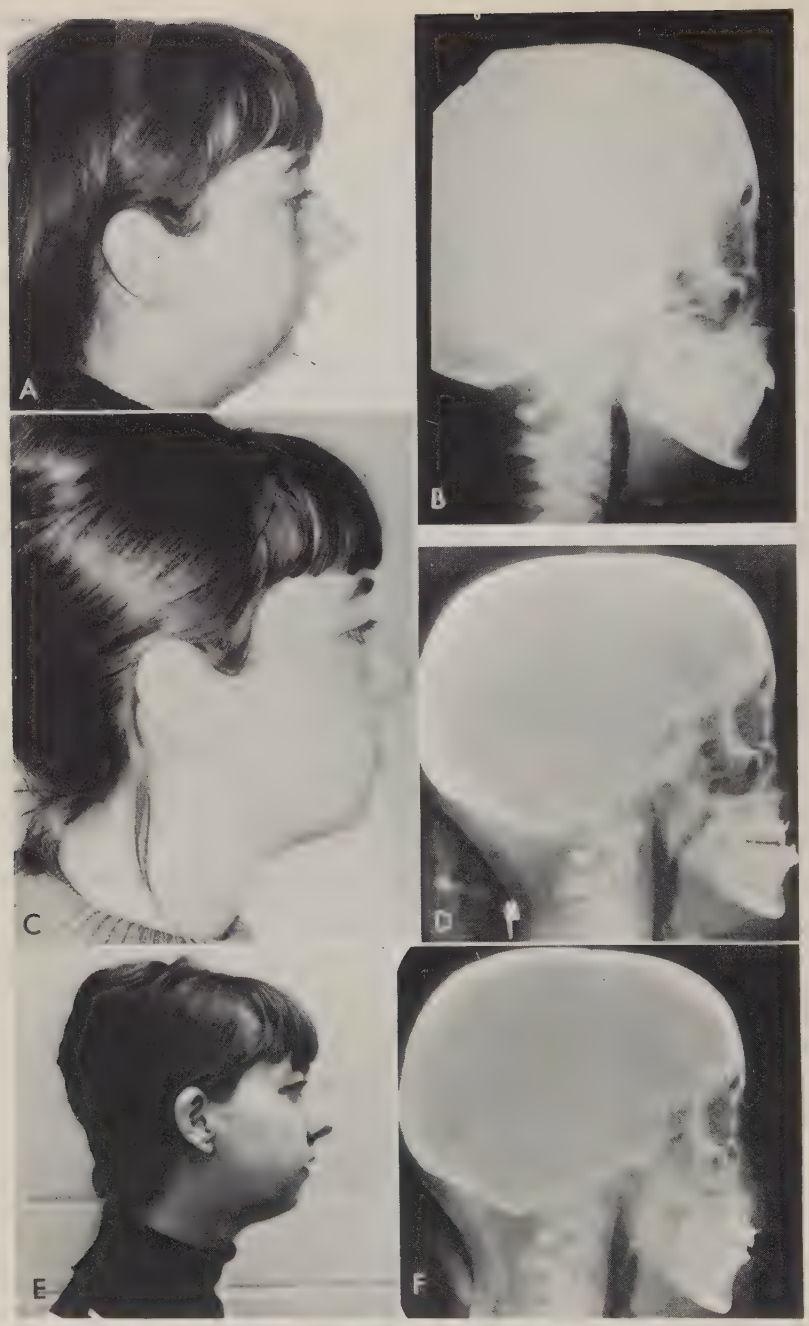

Figure 13. (Case 7). Complete bimaxillary retrognathia. *A, B,* The patient with a retrognathic profile but a normal occlusion according to the radiograph. Cephalometric analysis reveals both maxilla and mandible to be retrusive. *C, D.* First stage correction after positioning the mandible forward 10 mm. *E, F,* Second stage correction after moving the maxilla forward into the new occlusion.

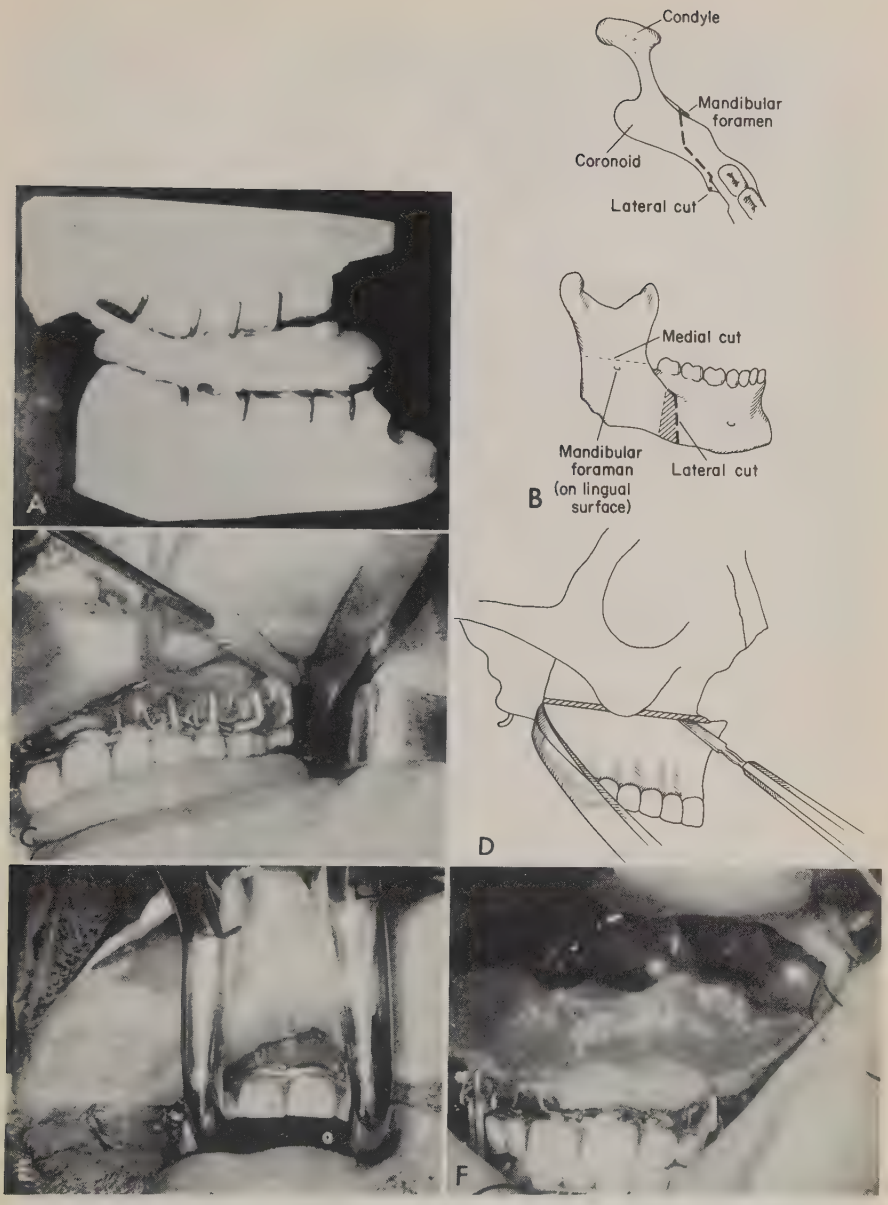

Figure 14. (Case 7). Complete bimaxillary retrognathia. *A*, Models, showing cast arch bars and an occlusal splint with a Class III malocclusion acquired by surgery. *B*, Drawings of the modified intraoral approach to the mandible of Obwegeser. *C*, *D*, LeFort I type surgery of the maxilla showing a small chisel separating the alveolar part of the maxilla from its superior portion and a larger chisel separating the tuberosity from the pterygoid plate. *E*, *F*, Disimpaction forceps in the nose and on the palate positioned for final separation of the alveolar maxilla.

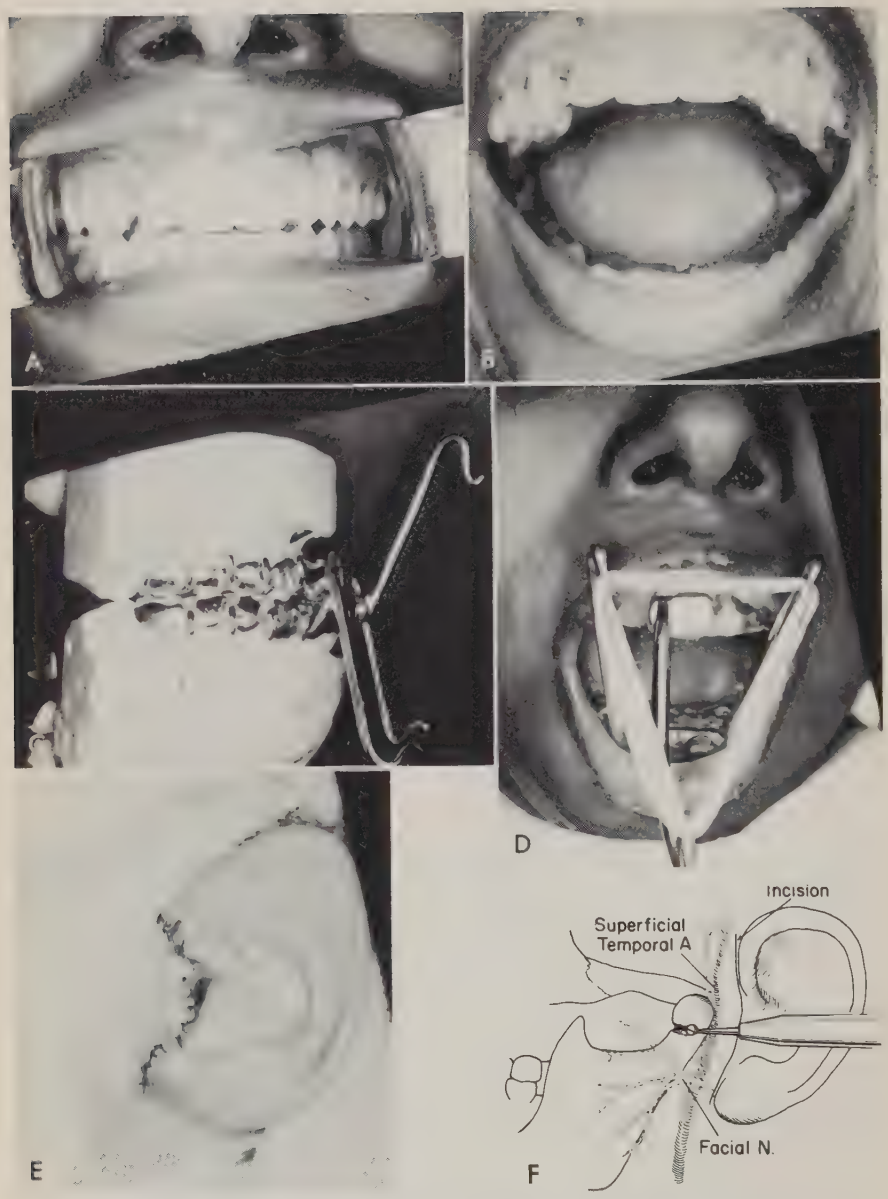

Figure 15. (Case 8). Bilateral temporomandibular joint ankylosis. *A*, One mm. opening of a patient with temporomandibular joint ankylosis due to rheumatoid arthritis. *B*. Thirty mm. opening immediately postoperatively. *C*. Cast metal splints and lugs for exercise elastics. *D*, Splints cemented to teeth and lugs activated with elastics. *E*, Preauricular line of incision. *F*, Drawing, modified from Henny,[19] showing surgery for a high condylectomy.

lateral walls are fractured, mobilizing the entire maxillary segment (Fig. 14*E* and *F*).

The maxillary segment is brought forward and placed into centric occlusion with the mandible. Bone grafts from the iliac crest are cut to form and placed over the lateral cuts in the area of the bicuspids and two blocks of bone are placed between the right and left maxillary tuberosities and pterygoid plates. The mucoperiosteum was closed and the patient placed in circumzygomatic suspension and intermaxillary fixation for another six weeks. The cephalometrics show an increase from SNA 74° to 80° in the maxilla and SNB 67° to 75° in the mandible, both of which are now within normal range. Figure 13*E* shows the patient 4 weeks after the second operation. The chin and neckline both show an aesthetic improvement and her occlusion is stable as it was in the preoperative position (Fig. 13*B* and *F*).

Modification of our procedure has been reported by several investigators.[8, 9, 31, 38] Developmental deformities seen in the Crouzon's and Apert's syndrome have been treated by clinicians[18, 28, 49] using modifications of the above technique as well as the LeFort type III osteotomy.

Case 8. Bilateral Temporomandibular Joint Ankylosis

This 22 year old woman had suffered from rheumatoid arthritis for 12 years. During the past 2 years, her temporomandibular joints had become progressively ankylosed. At the time of admission to the hospital,

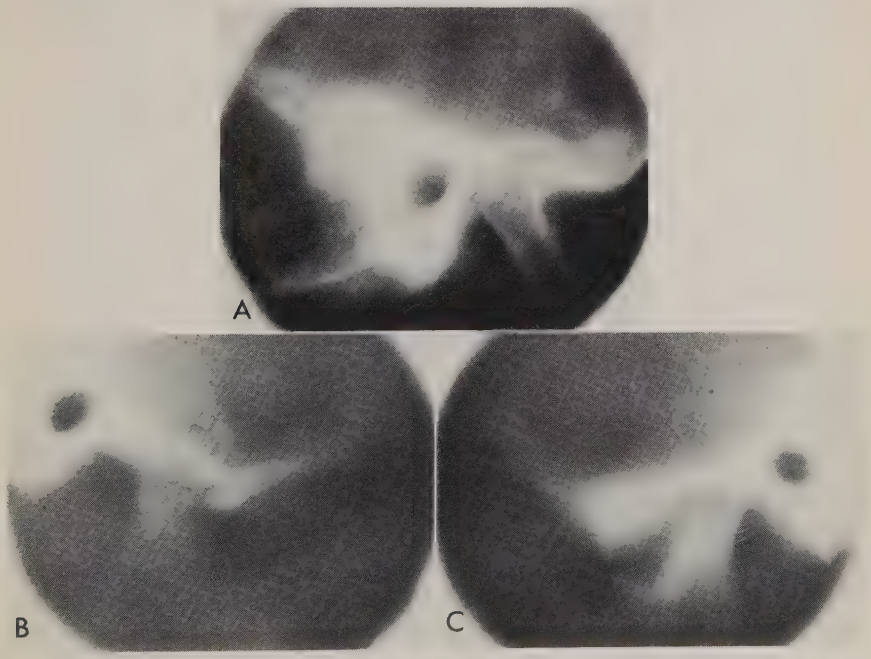

Figure 16. (Case 8). Bilateral temporomandibular joint ankylosis. *A*, A laminogram showing a normal right temporomandibular joint. *B*, The patient's right temporomandibular joint which was ankylosed but had a remnant of the meniscus remaining. *C*, The patient's left temporomandibular joint where complete bony ankylosis occurred, leaving no meniscus.

she could open her mouth only 2 mm. (Fig. 15*A*). She was losing weight. Radiographic laminograms reveal bilateral destruction of the mandibular condyles (Fig. 16*B* and *C*). Note the contrast to the normal temporomandibular joint (Fig. 16*A*).

Bilateral high condylotomies were the treatment of choice. A technique advocated by Henny[19] was used (Fig. 15*F*). A skin incision was made anterior to the ear at its superior and inferior attachments and along the posterior surface of the tragus (Fig. 15*E*). After the skin was reflected the incision was carried along the cartilage of the external auditory meatus down to the zygomatic arch. Here the condyle was palpated and the capsule entered. The right and left condyles were ankylosed to the temporal bone. Bilateral high condylectomies were performed. A portion of the right meniscus was present but none on the left side. A piece of Silastic was sutured over the left condylar stub and the incisions were closed. Postoperatively we were able to open the patient's mouth 30 mm. without difficulty (Fig. 15*B*). Many variations of this technique have been reported.[13, 14, 27, 39, 41, 45]

Impressions were taken, and cast metal splints were fabricated with removable arms on which elastic traction could be placed (Fig. 15*C*). Ten days later the castings were cemented and the patient was instructed on how to exercise (Fig. 15*D*). This therapy was continued for 3 months and the patient was counseled on how to continue the exercises after the removal of the castings. This was necessary to prevent further ankylosis and retropositioning of the mandible. Although the patient's generalized rheumatic pain continued, she began to eat better and her psychological outlook improved.

DISCUSSION

The individual medical problems of the cases presented here have not been mentioned. It should be pointed out, however, that all patients are carefully screened for any systemic disorder such as cardiac disease. For all patients blood is typed and cross matched, and two units of blood are available. All patients are treated with antibiotics and steroids before and after operation.

No surgical procedure is completely free of operative and postoperative complications. This is true in orthognathic surgery. A recent survey on complications has been presented by Gurnsey[17] and Behrman.[3a] Operative complications involve sectioning of the motor nerves, especially the marginal mandibular, buccal, and zygomatic branches of the facial nerve. Sensory loss may occur from damage to the terminal branches of the second division and the mandibular and mental branches of the third division of the trigeminal nerve. Control of bleeding is a necessary precaution, especially in the area of the facial vessels, the retromandibular vein, and the maxillary artery with its inferior alveolar, masseteric, and palatine branches. If an adequate blood supply is not maintained, there may be postoperative sloughing of entire bone segments, particularly in the maxilla. Loose and devitalized teeth are postoperative

hazards. Careful planning and surgery can prevent most of these complications.

As mentioned earlier a patient who is to have orthognathic surgery should be strongly motivated and be willing to take the time necessary for follow-up care. Relapse is a potentially constant postoperative complication, and the patient must be observed carefully especially during the time of fixation and followed for at least one year.

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